HANDBOOK of SURGERY

Weight Equivalents

Apothecary		Metric
1/320 gr	=	0 2 mg
1/210 gr	=	0 3 mg
1/160 gr	=	0 4 mg
1/120 gr	=	0 5 mg
1/100 gr	=	0 6 mg
1/60 gr	=	1 0 mg
1/30 gr	=	2 0 mg
1/16 gr	=	4 0 mg
1/12 gr	=	5 4 mg
1/10 gr	=	6 5 mg
1/8 gr	=	8 0 mg
1/6 gr	=	11 0 mg
1/4 gr	=	16 0 mg
1/3 gr	=	22 0 mg
3/8 gr	=	24 0 mg
1/2 gr	=	32 0 mg
3/4 gr	=	50 0 mg
1 gr	=	65 0 mg
1-1/2 gr	=	0 1 Gm
2 gr		0 13 Gm
3 gr		0 2 Gm
5 gr	=	0 32 Gm
7 1 2 gr	=	0 5 Gm
10 gr		0 65 Gm
15 gr		1 0 Gm
1 dr		4 0 Gm
1 oz		30 0 Gm

Volume Equivalents

Apothecary		Metric	
1 min	=	0 06	m
3 min	=	0 18	m
5 min	=	0 3	ml
8 min	=	0 5	ml
10 min	=	0 6	ml
13 min	=	0 75	ml
15 min	=	0 9	ml
16 min	=	1 0	ml
20 min	=	1 2	ml
30 min	=	1 8	
50 min	=	3 0	l
1 fl dr	=	3 7	ml
65 min	=	4 0	ml
80 min		5 0	
2 fl dr		7 5	l
2 2/3 fl dr		10 0	l
4 fl dr		15 0	l
5 1/2 fl dr		20 0	l
8 fl dr	=	1 0	fl oz
1 fl oz		30 0	m
1 2/3 fl oz		50 0	ml
2 fl oz		60 0	
3 3/8 fl oz		100 0	n
4 fl oz		120 0	
8 fl oz		240 0	
16 fl oz	=	480 0	

Household Measures		Apothecary		Metric
1 teaspoon	=	1 fl dr	=	4 ml
1 tablespoon	=	1/2 fl oz		15 ml
1 teacup	=	4 fl oz		120 ml
1 glass (tumbler)	=	8 fl oz	=	240 ml
1 measuring cup	=	8 fl z		240 ml
1 pint		16 fl oz		480 ml

CENTIGRADE TO FAHRENHEIT TEMPERATURES

C°	F°	C°	F°	C°	F°
35	= 95	37 5	= 99 5	40	104
35 5	= 95 9	38	100 4	40 5	104 9
36	96 8	38 5	= 101 3	41	105 8
36 5	= 97 7	39	= 102 2	42	= 107 6
37	98 6	39 5	= 103 1	43	109 4

MILLIEQUIVALENT CONVERSION FACTORS

To determine mEq. /L. of	Divide mg /100 ml or Vol % by
Calcium	2 0
Chlorides (from Cl)	3 5
(from NaCl)	5 85
CO_2 combining power	2 22
Magnesium	1 2
Phosphorus	3 1 (mM)
Potassium	3 9
Sodium	2 3

Preface

Our objective in preparing this Handbook has been to provide a concise summary of the essential features of the more common surgical disorders. We have tried to include basic diagnostic information and detailed discussions of nonoperative management, including preoperative and postoperative care, but in most cases the details of operative technic have been omitted. Exceptions to our rule about surgical technic have been made in the discussions of situations where the nonspecialist may need guidance in an emergency or in outlying areas where specialist assistance is not immediately available.

Space limitations have posed difficult decisions about what to include and the relative emphasis each discussion should receive. We do not suppose that all of our decisions have been the correct ones. Fortunately, new editions of the Handbook are scheduled every two years, on these occasions it will be possible to review each section and correct any disproportions or deficiencies which are found. The suggestions and criticisms of our readers will be of great assistance to us in this effort.

The editors wish to express their gratitude for the authoritative contributions of the men whose names appear in the Table of Contents. We wish also to thank Drs. Milton J. Chatton and Aubrey L. Abramson for their editorial assistance and many important contributions during all stages of preparation of the manuscript; Drs. Alyce Bezman, Ernest K. Gold, Albert Hall, William Heer, Judith Nadell, John Najarian, and Arthur M. Storment, Jr., for specific criticisms; Professor Ralph Sweet for his excellent illustrations; Mr James Ransom for editorial supervision of the project; and Mrs. Bernice Nelson for her expert secretarial assistance. But most especially we wish to thank our publisher, Dr. Jack D. Lange, for his initial conception of this book and for the creative spirit of collaboration with which he has directed all activities concerned with its publication.

<div align="right">

John L. Wilson
Joseph J. McDonald

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San Francisco, California
June, 1922

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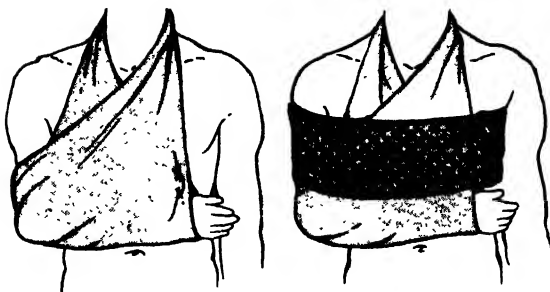
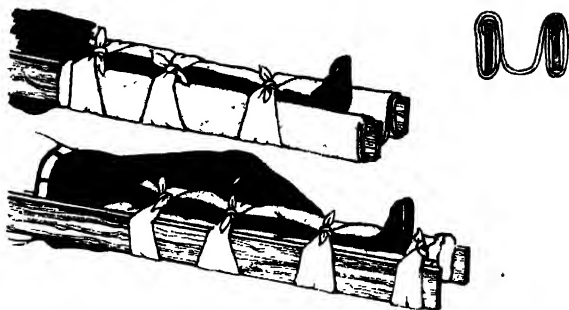Method of Application of Sling and Swathe
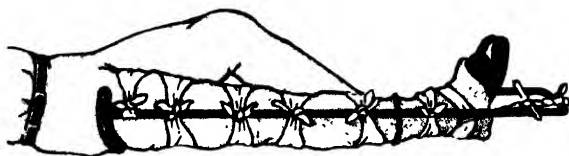
Keller-Blake Half-ring Splint for Transportation of Patient With
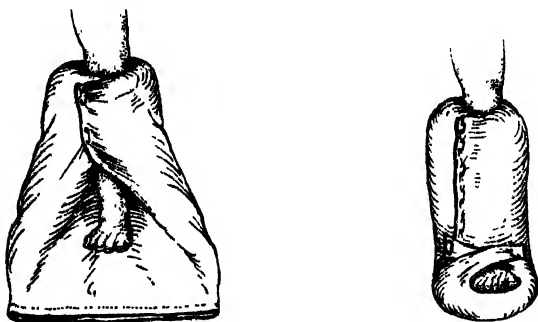Fracture of Thigh or Leg. Spanish windlass on a Collins hitch.
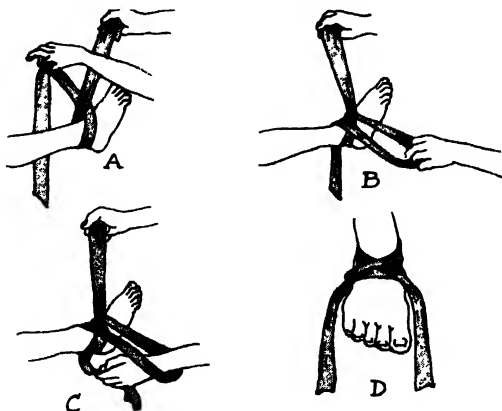
Method of Tying Collins Hitch

Padded Board Splints for Transportation of Patient With Fracture
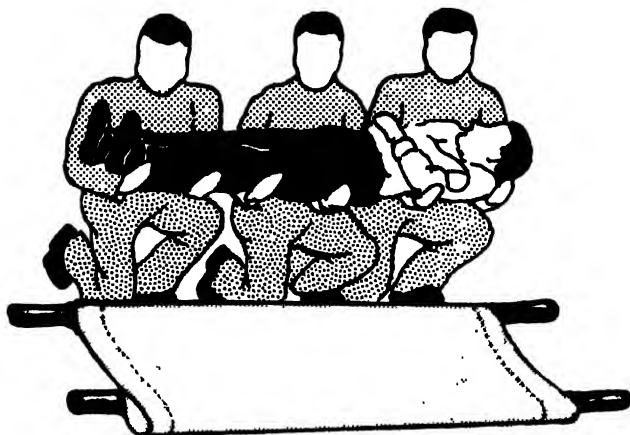of Thigh and Leg. Outer board extends to axilla.

Method of Application of Pillow Splints

Reinforcement of Pillow Splint for Transportation of Patient
With Injury of Ankle and Foot

Board or Door Used for Transportation of Patient
With Injured Spine

Method of Lifting Injured Patient Onto Stretcher

    a clean cloth and secure it firmly in place
B. Do not place antiseptic solutions or antibacterial powders in the
    wound   It is not necessary to cleanse the skin around the wound·
    with soap or antiseptic
C Arrange for the earliest possible cleansing, debridement, and
    closure under aseptic conditions, with anesthesia as indicated

Transportation of Injured Patients.
    Improper methods of moving patients can increase injuries
Lift severely injured patients with care (see above), and improvise
stretchers from blankets (see p. 4), boards, and doors when nec-
essary. Transport the following types of cases in the recumbent
position, on a stretcher. preferably in an ambulance: head and in·
ternal injuries; fractures of the spine. pelvis, and the long bones
of the lower extremities; shock; and major wounds in general. The
physician administering first aid is morally and in some areas le-
gally responsible for ensuring the transfer of the patient to a medical
facility or to another physician.

EMERGENCY ROOM WORK-UP

    Examine accident cases promptly and thoroughly. Record his-
tory and findings in accurate detail. Obtain the following informa-
tion in cases of acute injury.

Identification
  A Patient's name, address, phone number, age, sex, race, and
    marital status.
  B. Brought to emergency room by (give name and address).
  C. Referring physician (name and address).
  D. Next of kin (name and address)
  E. If injured at work, record the patient's occupation, the name,
    address, and phone number of his employer, and the name of
    the employer's insurance carrier.

History of Injury.
  A. Date and exact time of accident.
  B. Where and how it occurred.
  C. Accurate description of the mechanism of injury, including
    symptoms, subsequent course, and treatment given.
  D. In major injuries, take a complete general history.

Physical Examination.
  A In minor injuries, describe accurately the local findings. Line
    drawings are frequently helpful in showing the location and con-
    figuration of wounds.
  B. In major injuries, perform a complete physical examination
    Severe trauma commonly causes multiple injuries, and symp-
    toms may not become manifest for many hours   Palpate such
    patients carefully but completely in order not to overlook oc-
    cult fractures and visceral damage.

Laboratory Examination.
  A. In major injuries determine the Hct., Hgb., WBC, and dif-
    ferential count, and perform a urinalysis.
  B. Group and cross-match for blood transfusion as required.
  C. Obtain appropriate x-rays. All sites of suspected fracture
    should be x-rayed as soon as the patient's condition permits.
  D. Special laboratory tests as indicated.

Diagnostic Impression and Differential Diagnosis.

                    Signature of examiner
                    Date and hour of examination

Disposition.
  A. Discharge; date of return visit.
  B. Referral (name and address of physician).
  C. Hospital admission.

ARTIFICIAL RESPIRATION
(See drawings on back flyleaf.)

Begin artificial respiration immediately in any situation in
which breathing has stopped, and continue efforts at artificial res-
piration as long as the heart is beating. Artificial respiration by
mask or endotracheal tube is preferred when a mechanical resus-
citator or anesthesia apparatus is available.

Mouth-to-mouth Artificial Respiration.

This method has now superseded all others and is more effective than the Nielsen method.

Procedure. -

A. Do not delay. Every additional moment of apnea greatly reduces the chance of recovery.

B Clear the airway of foreign material by removing it manually or by patting the patient vigorously on the back in the prone position.

C Place the patient supine and kneel beside his head.

D Hold his chin and tongue forward in 1 of the following ways·
1. Insert your left thumb between the patient's teeth, grasp his mandible firmly at the midline, and draw it forcefully forward (upward) so that the lower teeth are leading.
2 Push strongly forward at the angle of the mandible

E Close the victim's nose with your right hand, or press your cheek against his nose.

F Take a deep breath, place your mouth firmly over the patient's mouth, and blow - forcefully in adults, gently in children. Watch the patient's chest. When it rises, take your mouth off his and let him exhale passively. Repeat inflations about 20 times per minute

Nielsen Method of Artificial Respiration.

When the mouth-to-mouth technic is not applicable, the next most effective procedure is the arm-lift-back-pressure method of Nielsen

SHOCK

Shock is a syndrome characterized by prostration and hypotension due to inadequate circulating blood volume

NEUROGENIC OR PRIMARY SHOCK
(Fainting)

This form of shock is due to a sudden outflow of autonomic impulses to the arterioles, producing vasodilatation or inhibition of constriction and rapid pooling of blood in the splanchnic bed and voluntary muscles   Cerebral blood flow is reduced and fainting occurs. Primary shock is usually caused by nervous or psychic stimuli, e.g., sudden pain, fright, fear, or vasodilator drugs (e g , nitrites).

Prodromal signs and symptoms are pallor, cold sweat, weakness, lightheadedness, and occasionally nausea. Fainting occurs abruptly thereafter, with transient hypotension and bradycardia.

Neurogenic shock is self-limiting. Rest in the recumbent or Trendelenburg position for a few minutes is usually sufficient. When faintness or prostration persists, other types of shock must be considered

HYPOVOLEMIC SHOCK
(Secondary, Oligemic, Hemorrhagic, Traumatic, or Surgical Shock)

In hypovolemic shock there is a true reduction in blood volume as a result of the escape of whole blood or plasma from the vascular space. Blood pressure is maintained initially by vasoconstriction, which reduces the size of the vascular bed. When this mechanism proves inadequate, hypotension results and tissue hypoxia increases. Prolongation of this state leads to irreparable damage to vital centers and shock becomes "irreversible." For this reason, early recognition of hypovolemic shock is imperative and treatment must be intensive.

Rapid loss of 15-20% of blood volume produces mild shock; 40% reduction causes severe shock; loss of over 50% of blood volume over a short interval is usually fatal. (Blood volume is approximately 7.5% of body weight)

Common Causes of Hypovolemic Shock.
A. Massive hemorrhage from wounds and internal injuries.
B. Severe crushing injuries, with extensive loss of blood and plasma into the tissues
C. Major fractures, especially of the proximal half of the femur. The mechanisms of blood and plasma loss are the same as in crushing injuries.
D. Extensive burns, with plasma loss and red cell hemolysis.
E. Nontraumatic hemorrhage, as from duodenal ulcer and esophageal varices; or reduction of plasma volume, as in severe peritonitis or dehydration with electrolyte imbalance.

Contributing Factors.
A. Age: The very young and the elderly tolerate loss of blood or plasma poorly.
B. Chronic Illness: The chronically ill often have a reduced blood volume. Relatively small diminution of circulating blood volume may cause failure of vascular compensation and precipitate shock. These patients can be identified by clinical acumen or, if available, blood volume determinations (see p 112).
C. Anesthesia: Paralysis of vasomotor tone by deep general or spinal anesthesia may precipitate shock in a patient who has previously compensated for a reduced blood volume.
D. Adrenal Insufficiency: Patients with Addison's disease suffer profound hypotension on minimal stress such as blood loss, trauma, or anesthesia. Patients who have within the previous 2 years been on regular therapeutic doses of corticosteroids (e.g., for arthritis or allergy) may have diminished adrenal cortical reserve and, unless supported by corticosteroids during and after surgery or other trauma, may develop resistant shock.

Clinical Findings.
A. Impending Shock: Before hypotension appears there is usually a period of vasoconstriction, with evidence of intense sympathetic activity. Weakness, pallor; cool, moist skin; and bradycardia or (usually) tachycardia are present. Hypovolemia should

♦

be suspected at this stage and treatment measures instituted. Do not confuse this with simple neurogenic shock.

B. Established Shock: Hypotension (systolic pressure below 90-100 mm. Hg) is superimposed on the above early signs, and tachycardia (over 100) usually develops. (In hypertensive patients, shock may be present when the systolic pressure is above 100 mm. Hg.) Thirst, air hunger, severe prostration, and dulling of the sensorium are advanced signs. Coma, cardiac arrest, and death are imminent at this point.

Preventive Measures.

Identify patients in whom shock is impending, and institute the following prophylactic steps without delay

A. Control and replace blood loss.

B Splint fractures.

C. Relieve pain and apprehension (see below)

D Keep patient recumbent.

E Avoid unnecessary movement. Rough handling and undue manipulation may precipitate shock

F. Treat all physiologic abnormalities which might intensify shock, e g., dehydration, electrolyte imbalance, and infection

Treatment.

Act rapidly. Shock is an acute emergency which takes precedence over all other conditions except gross hemorrhage and respiratory failure. Correction of hypovolemia by restoration of effective blood volume is the primary goal of antishock therapy.

A. Recumbent or head-down (Trendelenburg) position. Do not move the patient unnecessarily.

B. Maintain oxygenation. Clear the airway Rule out chest injury (see p 202). If dyspnea or cyanosis is present, administer oxygen by nasal catheter or mask (see p. 84).

C. Keep the patient comfortably warm. Excessive external heat produces undesirable vasodilatation

D Control pain promptly Use all appropriate first aid measures, including splints, dressings, and careful transportation. Shocked patients often have little or no pain When necessary, give morphine sulfate, 10-15 mg. ($1/6$-$1/4$ gr.) I.V. to ensure rapid action and to prevent delay in absorption from ischemic tissues. Do not give morphine to patients in coma or to those who have head injuries or respiratory depression

E Allay apprehension Reassure the patient A sedative is often more suitable than a narcotic. Use pentobarbital sodium, 0. 13 Gm. (2 gr.) subcut. or 0. 1 Gm. ($1^{1}/2$ gr.) orally.

F Parenteral Fluids: Restore adequate blood volume at once by the fluids immediately available. Whole blood is the most efficient fluid available for this purpose. Plasma, plasma products, plasma expanders, 5% dextrose, and normal saline are also effective, in that order

When starting the initial infusion, obtain blood for Hct., CBC, grouping, and cross-matching. When surface veins are unavailable because of peripheral collapse, it may be necessary to puncture the femoral vein for temporary infusion until a cut-down can be made in the antecubital space or ankle for insertion of a polyethylene catheter.

1 Whole blood - When whole blood has been lost, use proper-
ly grouped and cross-matched whole blood for replacement
if possible. In severe shock after extensive trauma, it may
be necessary to transfuse massively. Transfusion of over
4000 ml in one-half hour has been required for resuscita-
tion If overloading is feared, observe venous pressure
carefully and give blood until the systolic pressure is above
100 mm. Hg or venous pressure is above 15 cm water.
Group O (universal donor) blood may be used without cross-
matching if necessary to save life Pressure transfusion
through multiple venipunctures may be advisable. Intra-
arterial transfusions have special value only when cardiac
action has become markedly depressed

2 Plasma, serum albumin, or Plasmanate® - Plasma and its
products can be stored and made immediately available for
emergencies They are specifically indicated when plasma
loss has led to hemoconcentration, as in burns and perito-
nitis Pooled plasma is most likely to transmit homologous
serum jaundice and should therefore be used only when other
agents are inadequate or unavailable Normal Human Serum
Albumin, U S P., or salt-poor serum albumin contains 25
Gm. albumin/100 ml. and is equivalent in osmotic pres-
sure to 500 ml. of plasma It is very expensive. Plasman-
ate® is a solution of albumin, alpha-globulin, and beta-
globulin These proteins are obtained as a by-product dur-
ing the Cohn fractionation of human serum in the process of
recovering gamma globulin and fibrinogen Since its os-
motic activity is comparable to that of plasma, it is quite
effective in maintaining blood volume. Plasmanate® and
serum albumin will not transmit homologous serum jaun-
dice

3. Plasma expanders - These agents are solutions of inert or-
ganic molecules of high molecular weight and size They are
lost slowly from the vascular compartment and thus serve to
increase plasma volume The only one which can be recom-
mended is Dextran, N N D (Expandex®, Gentran®, Plavo-
lex®), a water-soluble biosynthetic polysaccharide, 6% in
isotonic saline solution. Administer 500-1000 ml. I.V. at
a rate of 20-40 ml. /minute Use cautiously in patients
with cardiac or renal insufficiency to avoid pulmonary edema
Anaphylactoid reactions have been reported Because ad-
ministration of dextran may interfere with tests for blood
grouping and cross-matching, blood samples for these ex-
aminations should be drawn beforehand In order to avoid
hemodilution, the dosage should not exceed that which will
- maintain systolic blood pressure at 85-90 mm Hg.

4. Saline and glucose - These fluids will expand blood volume
for an hour or so until blood or plasma can be obtained Give
500-1000 ml. of physiologic saline, 5% dextrose in water, or
5% dextrose in saline while awaiting other fluids.

G. Vasopressor Drugs: These agents are of greatest value in the
management of hypotension not associated with decrease in
blood volume In hypovolemic shock, vasopressor drugs should
not be used in lieu of fluids which will expand the blood volume
Nevertheless, they are occasionally valuable adjuncts to

treatment before the arrival of blood or plasma. At such
critical times a vasopressor will frequently sustain BP tem-
porarily and preserve circulation to vital centers. In rare
instances of advanced shock the administration of blood or
plasma must be supplemented with a vasopressor to raise the
BP. Dosage levels (see p. 623) for the various agents are
empirical and must be adjusted to obtain the desired BP re-
sponse.

Evaluation of Antishock Therapy.

Constant observation is essential. Accurate determination of
blood volume deficit is rarely feasible in an emergency. Fluid re-
quirements must be estimated clinically, and the response of vital
signs is the most dependable guide to therapy. When systolic BP
rises above 100 mm. Hg, tachycardia lessens, and the skin be-
comes warm and dry, shock is abating and infusions may be slowed
or stopped while progress is observed.
  A. Record BP, pulse, and respirations every 15-30 minutes.
  B. Measure fluid intake and output. Severe or prolonged shock
     may produce acute renal failure (see p. 43).
  C. Hct. and CBC should be repeated at appropriate intervals for
     comparison with original baseline values   (Keeping in mind
     the effect of hemodilution and hemoconcentration.) Determine
     blood volume by the radioisotope or dye-dilution technic if
     possible and repeat as needed.
  D. Listen to the patient's chest for signs of pulmonary edema and
     observe venous pressure if excessive fluid administration is
     feared.

Persistent or Recurrent Shock.

The cause of persistent shock must be sought immediately. If
shock does not relent within 30-60 minutes, one of the following
mechanisms may be present:
  A Inadequate Blood Volume:
     1. Inadequate blood replacement - Transfusions must fre-
        quently be rapid and massive, particularly after extensive
        trauma.
     2. Continued blood loss.
     3. Failure to use vasopressors and/or corticosteroids in pro-
        found, resistant shock.
  B. Severe Injury:
     1. Ruptured viscus.
     2. Thoracic injury interfering with circulation, e g., cardiac
        tamponade, tension pneumothorax, flail chest (see p. 202).
     3. Brain injury or hypoxic damage
  C. Coincident disease, e.g., heart disease, infection, electrolyte
     imbalance.
  D. "Irreversible" shock due to prolonged tissue hypoxia

SEPTIC SHOCK

Overwhelming infection may cause circulatory collapse with
hypotension due to profound toxemia, vasodilatation, and direct
toxic action on the heart and adrenals. Serious infection may be

unsuspected or may be obscured by antibiotic therapy. Shock in a febrile patient, if hemorrhage or other obvious cause is not present, should be considered septic in origin and treated accordingly.

Diagnostic Features.
The patient is febrile   There may or may not be a known infection. No other cause for shock is present. A high WBC (20,000 or more) is usually found, and the differential count usually shows a shift to the left. Hemoconcentration and oliguria are common findings. Response to parenteral fluids alone is poor.

Treatment.
The objectives are to raise and maintain BP while instituting measures to combat infection.
  A. Vasopressor drugs are often essential to raise BP above 100 mm. Hg (see p. 623).
  B. Parenteral fluids should consist of plasma, if hemoconcentration is present; or whole blood, if anemia is present. Dehydration and electrolyte balance must be corrected (see p. 99).
  C. Give intensive specific antibiotic therapy if the infectious agent is known   If the organism is not known, take 2 or 3 blood cultures within a few hours and repeat every 12 hours and when temperature spikes or chills occur. Empiric antibiotic administration must be considered carefully   Localized infections must be sought for and drained.
  D. Corticosteroids (see p. 607) may be necessary to combat severe intoxication and hypotension in an effort to preserve life until sepsis is controlled
  E. General supportive measures include the administration of oxygen and the treatment of accompanying conditions. As in hypovolemic shock, urine output must be maintained by adequate BP and fluid intake   An indwelling catheter may be essential for accurate follow-up of renal function.

CARDIOGENIC SHOCK

Cardiogenic shock may be described as a failure of the circulation resulting from acute decrease of cardiac output (so-called "forward heart failure"). It is typically characterized by severe hypotension, low pulse pressure, oliguria, cold and clammy skin, and dulling of the sensorium. It may be caused by myocardial infarction, cardiac tamponade, severe tachycardia, terminal congestive failure, or massive pulmonary embolism. Recognition of cardiogenic shock is important since intensive parenteral fluid administration, as for hypovolemic shock, is usually contraindicated. Treatment consists of relief of pain, administration of oxygen, correction of arrhythmia; administration of cardiac drugs such as digitalis when indicated, relief of tamponade if present, and control of hypotension with vasopressors (see p. 623). Cardiogenic shock may be precipitated by trauma or may be an intercurrent development in the course of a surgical illness.

ANAPHYLACTIC REACTIONS
(Anaphylactic Shock)

These catastrophic and frequently fatal allergic reactions may occur within seconds or minutes after parenteral administration of animal sera or drugs, including antibiotics. Although there is occasionally no history of previous exposure to the foreign substance, anaphylaxis undoubtedly represents induced hypersensitivity by previous ingestion or injection.

Clinical Findings.
Symptoms and signs include apprehension, generalized urticaria or edema, a choking sensation, wheezing, cough, or status asthmaticus. In severe cases there may be hypotension, loss of consciousness, dilatation of pupils, incontinence, and convulsions, with death occurring suddenly.

Emergency Treatment. (Act immediately!)
A. Position the patient for comfort and ease of respiration If he is unconscious, place him in the supine or slightly head-down position
B. Maintain oxygenation and keep the airway open (see p. 84). If respirations have ceased, give artificial respiration by the mouth-to-mouth technic (see p 7) or with mask and positive pressure oxygen breathing (see p 84).
C. Medical Measures: Epinephrine is the drug of choice for emergency use It may be necessary to supplement it with intravenous antihistaminic drugs or steroids and, in case of intense bronchospasm, with intravenous aminophylline (see below).
   1. Epinephrine Hydrochloride, U.S.P. - (The drug of choice.)
      a. I.M. - 1 ml. of 1:1000 solution I M.; repeat in 5-10 minutes and later p r.n.
      b I.V. (for even more rapid effect) - 0 1-0 4 ml. of 1:1000 solution in 10 ml. of saline slowly I.V.
   2. Diphenhydramine Hydrochloride, U.S.P. (Benadryl®), or Tripelennamine Hydrochloride, U.S.P. (Pyribenzamine®), 10-20 mg. ($1/6$-$1/3$ gr.) I.V. if response to epinephrine is not prompt and sustained.
   3. Hydrocortisone Sodium Succinate, N.N.D. (Solu-Cortef®), 100-250 mg. I.V. over a period of 30 seconds as adjunct to epinephrine and diphenhydramine. Dosage depends upon the severity of the condition; the drug may be repeated at increasing intervals (1, 3, 6, 10 hours, etc.) as indicated by the patient's clinical condition
   4. Aminophylline Injection, U.S.P., 0.25-0 5 Gm. ($3^{3}/4$-$7^{1}/2$ gr.) in 10-20 ml. saline I.V. slowly for severe bronchospasm. Duration of action is 1-3 hours. May repeat in 3-4 hours.

Prophylaxis.
Avoid using potentially dangerous drugs or sera if possible. Be particularly cautious when administering parenteral medications to patients with a history of allergy or previous reaction to the agent to be injected. Give injections slowly and keep such individuals under close observation for an hour or more thereafter as required.

Always perform a sensitivity test (intradermal or conjunctival) before injecting animal sera or other agents to which a hypersensitivity reaction may occur. When sensitivity is demonstrated by a positive test or is suggested by the history, the patient must be desensitized by the administration of a series of divided doses.

COMA

Coma is loss of consciousness from which the patient cannot be aroused. A patient is considered semicomatose if he can be partially aroused by loud commands or painful stimuli but promptly lapses into unconsciousness again when the stimulus is withdrawn. Coma may be caused by poisoning (e g., alcohol, barbiturates, narcotics, carbon monoxide), cerebral lesions (e g., trauma, vascular accidents, tumors, infections, epilepsy), metabolic and constitutional disorders (e.g., diabetes mellitus, hypoglycemia, Addison's disease, uremia, hepatic coma, eclampsia), and by such miscellaneous disorders as toxemia of infection, hypovolemic shock, asphyxia, heat stroke, severe heart failure, and hysteria.

Diagnosis.
The "diagnosis" of coma consists of determining the specific causative factor This requires a searching history from the patient or his friends or relatives and a complete physical examination with specific attention to the neurologic examination. When the diagnosis is still in doubt, the following may be of value: urinalysis, CBC; blood glucose, urea, ammonia, electrolytes, and alcohol; CSF examination, skull x-rays, and blood cultures.

Treatment.
  A. Emergency Measures: Recognize and treat life-endangering conditions immediately.
     1. Maintain adequate oxygenation. This is a cardinal principle in all emergency treatment.
        a. Keep the airway clear.
        b. Use artificial respiration if breathing is failing or has stopped (see p. 6).
        c. Administer oxygen by nasal catheter, mask, or tent if oxygenation is poor, as indicated by depressed respirations, cyanosis, or dyspnea.
     2. Treat shock.
     3. Hospitalize the patient as soon as possible.
  B. General Measures: Observe the patient constantly, record vital signs at regular intervals, and change his position every 30-60 minutes to avoid hypostatic pneumonitis and decubiti. A lateral and slightly head-down position is best for patients likely to vomit. Have a suction machine and an alert attendant near the bedside.

     Maintain fluid, electrolyte, and caloric intake (see pp. 86 and 99). Consider tube feeding if coma lasts more than 2-3 days (see p. 91). Recording the urine output is the best guide to fluid therapy (see p. 101). Catheterize if the bladder is

distended or if the patient is unable to void within 8 hours. In critically ill patients and those with urinary retention, an indwelling catheter is advisable for closer observation of urine volume.

Avoid narcotics, sedatives, and other medications until the diagnosis is established; marked restlessness can then be treated by administration of a parenteral sedative or tranquilizer preparation (see pp. 607 and 622).

DIAGNOSTIC FEATURES OF SPECIFIC TYPES OF COMA

Nonsurgical causes of coma must be promptly recognized when they occur in surgical patients. Trauma and surgical operations not infrequently aggravate a preexisting metabolic or other derangement and cause rapid deterioration and coma. Medical consultation should be sought immediately.

Acute Alcoholic Intoxication.

Always rule out other causes of coma, especially head trauma. Diagnosis is based on a history of drinking, alcoholic breath, flushed face, injected conjunctivas, slow and stertorous respirations, diminished reflexes, and a blood alcohol level above 0.5% If coma is light, it may alternate with periods of delirium and restlessness.

Narcotics Poisoning.

Small doses of narcotics may cause respiratory depression and coma in liver insufficiency, myxedema, emphysema, head injuries, and in debilitated or elderly patients. Delayed absorption of toxic amounts may occur if multiple subcut. or I.M. injections are given during a period of vasoconstriction, as in hypovolemic shock.

Diagnostic features include cold, clammy, cyanotic skin; pinpoint pupils, respiratory depression (breathing slow and irregular, sometimes Cheyne-Stokes); and a feeble and often irregular pulse

Diabetic Coma.

Coma may be precipitated in a diabetic patient by infection or failure to regulate insulin dosage. Diagnostic features include the following: history of diabetes, gradual onset with blurred vision and thirst, air hunger or Kussmaul breathing, dehydration (soft eyeballs), acetone breath ("fruity" odor on breath), glycosuria, acetonuria, hyperglycemia, ketonemia, and low plasma bicarbonate.

The objective of treatment is to correct the acidosis by administering large doses of insulin with an abundance of fluid to overcome dehydration. The greatest single factor in reducing mortality is vigorous insulin therapy during the first 2-3 hours. Act promptly when coma is impending or present, and obtain the consultation of an internist.

Diabetic acidosis can be prevented by careful regulation of insulin and caloric intake during surgical illnesses (see p. 45).

Hypoglycemic Shock.

Weakness, hunger, irritability, faintness, tremors, and convulsions or coma. History of diabetes and use of insulin. Blood sugar consistently below 50 mg./100 ml. No sugar in urine.

If the patient is conscious, give sugar, glucose, or sweetened orange juice by mouth. If he is unconscious, give glucose I.V. (preferably 50%).

Uremic Coma.

A history of chronic renal disease may be obtained The breath is urinous, and there is twitching of the extremities and, frequently, convulsions Albuminuric retinitis, papilledema, and hypertension are characteristic of severe chronic nephritis. Urinalysis shows low sp. gr. (about 1.010), albumin, cells, and casts. Blood NPN is usually over 100 mg./100 ml., and potassium levels are elevated. Blood bicarbonate and pH are depressed.

BLAST AND CRUSH INJURIES

BLAST INJURY

The blast force from an explosion may be transmitted to the body through air, water, or solid objects on which the patient is standing or resting. Serious internal damage may occur without evidence of surface injury. Victims are most likely to suffer damage or rupture involving the lungs or abdominal viscera. Localizing symptoms and signs are frequently delayed in onset.

Widespread alveolar hemorrhage with dyspnea, frothy hemoptysis, and cyanosis is typical of blast injury to the lungs. Tracheostomy may be required to aspirate secretions and administer oxygen (see p. 84). Do not overlook pneumothorax (see p. 204) or hemothorax (see p 203). Mild contusions of the abdominal viscera cause moderate discomfort and occasionally colicky pain which subsides within 48-96 hours. More serious injuries with impending or actual perforation produce signs of peritoneal irritation or shock (see p. 1).

Rupture of the ear drum by blast causes severe pain and often deafness. No local treatment is required. Packs and ear drops are contraindicated.

CRUSH INJURY

Patients who have been crushed and whose limbs have been compressed for an hour or more are likely to develop hypovolemic shock due to extravasation of blood and plasma into the injured tissues after the compression is released. If this shock is not treated, the prolonged hypotension often causes kidney damage and acute renal insufficiency. The entire clinical picture of compression injury, shock, and acute renal failure is known as the "crush syndrome."

Treatment consists of controlling shock (see p. 7) and observing carefully for acute renal insufficiency (see p. 43).

BURNS

Burns are caused by a wide variety of agents, including flame, hot water, steam, electricity, and chemicals. The general principles of treatment are the same in all.

Depth or Degree of Burn.
  A. First Degree: Erythema.
  B Second Degree: Erythema with blistering.
  C. Third Degree: Destruction of full thickness of skin and frequently of deeper structures also

Estimate of Extent of Burn.
    The amount of body surface burned and the depth of the burn determine the fluid losses. The "rule of nines" is a useful means of estimating the percentage of total body surface involved by second or third degree burns of specific skin areas (see p. 19). Each upper extremity and the head are considered to represent 9% of the total surface area; each lower extremity, 18%; the anterior surface of the trunk, 18%; and the posterior surface of the trunk, 18% Second or third degree burns of over 20% of total body surface usually cause marked fluid loss which results in burn shock. The mortality rate in second or third degree burns of 50% of total body surface is about 50%; second and third degree burns of over 75% of total body surface are almost always fatal.

Special Laboratory Examinations.
  A. Hematocrit: In severe burns, determine the Hct. repeatedly as a guide to fluid therapy.
  B. Blood Typing and Cross-matching: Blood transfusions are usually indicated in extensive burns.

Replacement Solutions.
    Fluid and electrolyte replacement by the oral route can frequently be employed, alone or as a supplement to the intravenous route. For oral administration, a solution containing 3 Gm. /L of sodium chloride and 1.5 Gm. /L. of sodium bicarbonate may be used. This mixture contains 70 mEq. /L. of sodium; it is hypotonic and therefore better tolerated than isotonic solutions.
    For intravenous use a balanced electrolyte mixture such as Lactated Ringer's Injection, U.S.P., which contains a mixture of sodium chloride and sodium lactate, is preferred to normal saline. Treatment of shock requires the use of blood and either plasma or a plasma expander such as Dextran, N.N.D. (Expandex®, Gentran®, Plavolex®).

Symptoms of Fluid Deficiency in Burns.

Very close attention to clinical signs and symptoms is of great importance, particularly during the first 24 hours after the burn has occurred Excessive thirst, vomiting, restlessness, disorientation, and mania - together with increase in pulse rate, decrease in BP, collapsed veins, and oliguria - are indications that fluid losses have exceeded the rate of fluid replacement. During this critical early phase the adequacy of treatment is best judged by the urinary output, which should ideally be 30-50 ml /hour. However, if the rate of urinary excretion is below these suggested volumes, it is important to exclude acute renal insufficiency as a cause of the oliguria before increasing the fluid intake. The diagnostic criteria for the occurrence of acute renal insufficiency are given on p. 43.

Urine volumes greater than 100 ml /hour indicate that too much fluid has been given, but after 48 hours the urinary output is completely unreliable as a guide to therapy. In part this is due to the release of nitrogenous wastes from the burned tissues, which act as diuretics, in addition, electrolyte deficits may force compensatory elimination of water, as in the developing phase of a low-salt syndrome. Under these conditions, therapy is guided almost exclusively by clinical signs and symptoms, using enough fluid to maintain normal turgor of the unburned skin, fullness of the veins, and moisture of the oral mucosa. The quantities of fluid required to accomplish this may be surprisingly large. However, care must be taken to avoid overhydration and consequent water intoxication, which produce edema of the unburned tissues and, in severe cases, coma and death.

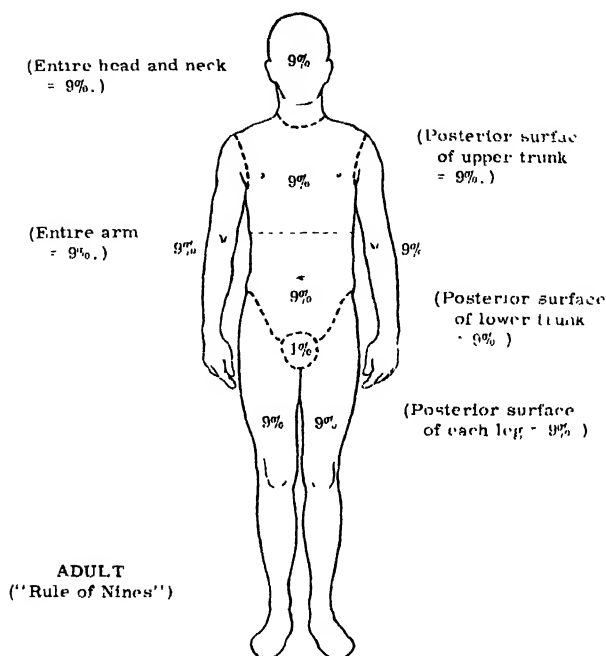### Calculation of Replacement Therapy.

As a general rule, burns involving less than 15-20% of the body surface in adults do not require the use of solutions other than saline or water given orally and/or intravenously. More extensive burns require very careful attention to fluid intake and output. It is therefore recommended that a 15-18 gauge cannula or plastic tube be firmly secured in a vein and that an indwelling catheter be placed in the bladder.

The severely burned patient requires, in the first 24 hours, approximately 1 ml. of blood (or plasma or a plasma expander) and 1 ml of a balanced electrolyte solution for each 1% body surface burned/Kg. body weight In addition, adults require 1000-2000 ml. of either an electrolyte-containing solution or water. This relationship of fluid requirement to involved surface area and body weight is not linear; therefore, if more than 50% of the body surface is burned, the calculations should be based only on a 50% involvement In no case should more than 10,000 ml of fluid be given in the first 24 hours. During the second 24 hours, reduce the fluid intake to one-half to three-quarters of that given in the first 24 hours. In the third 24 hours, shift to oral intake.

Example:
1. 70 Kg man with a 30% burn.
2. Blood and/or plasma or plasma expander = $1 \times 70 \times 30$
   = 2100 ml.
3. Electrolytes (e g , Lactated Ringer's Injection, U.S.P.)
   = $1 \times 70 \times 30$ = 2100 ml.

ESTIMATION OF BODY SURFACE AREA IN BURNS

(Entire head and neck = 9%.)

(Posterior surface of upper trunk = 9%.)

(Entire arm = 9%.)

(Posterior surface of lower trunk = 9%)

(Posterior surface of each leg = 9%)

ADULT
("Rule of Nines")

Variations from Adult Distribution in Infants and Children (in %)*

	New-born	1 yr.	5 yrs	10 yrs
Head	19	17	13	11
Both thighs	11	13	16	17
Both lower legs	10	10	11	12
Neck	2			
Anterior trunk	13			
Posterior trunk	13			
Both upper arms	8	These % remain		
Both lower arms	6	constant at all		
Both hands	5	ages.		
Both buttocks	5			
Both feet	7			
Genitals	1			
	100			

*After Berkow.

4. Glucose in water or Lactated Ringer's Injection, U.S.P,
    = 2000 ml.
5. Total fluids first 24 hours = 6200 ml.
6. Give one-half (3100 ml.) in the first 8 hours, one-
    fourth (1550 ml.) in the second 8 hours, and one-
    fourth (1550 ml.) in the third 8 hours.

Criteria for Use of Whole Blood or of Plasma.

When the Hct. is high, whole blood cannot be used unless
large quantities of electrolyte-containing solutions are given si-
multaneously. If the Hct. is below 60% and is decreasing, whole
blood may then be used; on the other hand, a Hct. above 60% which
is increasing indicates a primary need for electrolyte-containing
solutions and/or plasma or a plasma expander.

It is expected that blood will be needed in patients whose burns
are deep and extensive or who show signs of peripheral circula-
tory collapse if at the same time the Hct. is low and electrolytes
alone have failed to bring about clinical improvement

Use of Potassium.

The excretion of potassium is high during the acute phase of
burns and may remain elevated for several weeks In addition, a
poor food intake at this time will prevent adequate replacement of
potassium. Beginning on the third or fourth day of treatment, give
potassium chloride, 3-4 Gm. (45-60 gr) orally in fruit juice or
broth 3 times a day until a full normal diet is taken.

Additional Guides to Fluid Therapy

A Water Tolerance Test: When it is suspected that low urine
   output is due to inadequate intake rather than to renal failure,
   give 1000 ml. of 5% dextrose in water I.V. in one hour. A
   sharp rise in urine output during or immediately after the in-
   fusion suggests that the kidneys are functioning satisfactorily
   and that fluid intake should be increased
B. Phenol Red (PSP) Test: If hydration seems adequate but urinary
   output is abnormally low, administer PSP, 1 ml I.V Excre-
   tion of more than 10% of the dye in one-half hour indicates that
   acute renal failure is not present and that oliguria is due to in-
   adequate fluid intake.

EARLY CARE OF THE BURN WOUND

First Aid.

A. First degree burns require no treatment.
B. Minor second degree burns may be washed carefully with bland
    soap and water and dressed with sterile petrolatum and gauze
    and a pressure bandage. Change the dressing after 5-8 days.
C. Severe burns should not be washed, greased, powdered, or
    painted with medications of any kind. Wrap the burned area in
    clean towels or sheets and transfer the patient to a hospital im-
    mediately.

Surgical Measures in Severe Burns.

A. Control pain, which is usually marked, with morphine sulfate,

10-15 mg. ($1/6$-$1/4$ gr.) I.V. or I.M., or other narcotic (see p. 605). General anesthesia is rarely necessary for the initial cleansing and dressing of severe burns if a narcotic is used and procedures are done gently

B Treatment of Burned Area:

1. Aseptic technic is essential. Wear cap, mask, sterile gown, and gloves when dressing burns. Sterile linen, instruments, and dressings are required.

2 Cleanse burn and surrounding area with bland or hexachlorophene soap and sterile warm water. Wash gently with gauze sponges   Remove grease or oil with ether or benzene

3. Debride carefully. Remove only loose and necrotic tissue. Puncture blebs aseptically and leave them in place as protective coverings

4 Apply petrolatum gauze and a pressure dressing. Place a single layer of petrolatum (or Xeroform®) gauze smoothly over the burn, cover this with soft pads or other absorbent dressings, and secure firmly in place with stockinet or elastic or gauze bandage. The application of antibiotics and chemotherapeutic agents to the surface of extensive burns is contraindicated because of the danger of toxic or sensitivity reactions

5 Exposure treatment - In this form of treatment, no dressings are applied to the burn after cleansing and debridement. On exposure to air a coagulum of serum seals the burn wound. This is the preferred method of treating burns of the head, neck, genitalia, and perineum. It is also suitable for limited burns on one side of the trunk or extremity. In mass casualties it may be necessary to treat extensive burns in this manner. The patient is placed on clean sheets and turned frequently when burns encircle the body in order to avoid maceration   If infection occurs beneath it, the coagulum should be removed and warm saline compresses applied to the area.

C. Prevention of Infection: Reliance is placed on thorough cleansing of the burn and on aseptic dressing technics. Prophylactic antibiotics are rarely used. Cultures are obtained from exudates, and specific antibiotics chosen on the basis of sensitivity studies when signs of infection appear.

D. Immunization against tetanus (see p. 130) should be given during the first 24 hours in all major burns.

Burns of Specific Anatomic Areas.

A. Respiratory tract burns should be suspected whenever extensive burns of the head and neck occur   Inhalation of flame or hot gases produces severe tracheobronchitis and pneumonitis. Obstructive laryngeal edema may develop rapidly, preceded by stridor, copious respiratory tract secretions, dyspnea, and cyanosis. Tracheostomy should be done without delay if there is significant obstruction or retained secretions. It is justifiable to give penicillin, 1 million units, and streptomycin, 0.5 Gm., every 12 hours empirically in respiratory tract burns until sputum cultures can be obtained and specific antibiotics chosen.

B. Head and neck burns are treated by exposure (see above). They

are often less deep than suspected initially, and rapid healing is favored by the great vascularity of the region. Early grafting of eyelid burns is especially important to avoid ectropion and corneal ulceration due to exposure.

C. Hand burns (see also p. 475) must be carefully cleansed and the fingers dressed individually with petrolatum gauze. Remove rings. Immobilize the entire hand in the position of function by pressure dressings and splints. Soon after the first redressing, which is done 5-8 days after the burn, areas of third degree burn should be excised in a bloodless field (using a pneumatic tourniquet) and a skin graft applied in order to obtain the earliest possible restoration of function.

D. Joint areas should be maintained in optimal position and all third degree involvement grafted early to avoid disabling contractures.

E. Perineum and genitalia burns are left exposed and cleansed with soap and water when they become soiled with feces or urine An inlying Foley catheter for constant drainage of the bladder may be advisable in genital burns.

LATER CARE OF THE BURN WOUND

Re-dressing and Reevaluation.

Observe strict aseptic technic in all burn dressings. Remove the original burn dressing down to the petrolatum gauze after 5-8 days. The depth and extent of the burn can be accurately determined at this time. Second degree burns require only reapplication of the pressure dressing and should heal in about 2 weeks. Third degree burns demand special management.

Treatment of Third Degree Burns.

A. Removal of Slough: The necrotic surface of a third degree burn usually does not separate for many weeks. Significant areas of slough or necrosis should therefore be carefully removed in the operating room under general anesthesia 10-14 days after the burn if the patient's general condition allows. Burns of the face permit more conservative debridement since slough separates rapidly in this region.

B. Skin Grafting: (See p. 439.) Early skin grafting (preferably within the first few weeks following the burn) is essential to avoid chronic sepsis, malnutrition, and scar contractures. Skin grafting should be started as soon after removal of slough as possible. The denuded granulating surface should be firm and bright red, with a minimum of exudate. Warm saline dressings (changed several times daily) may be of great assistance in the final preparation of the burn wound for skin grafting.

C. Control of Infection: Signs of infection include rising temperature, tachycardia, general toxicity, local pain and tenderness, and increased drainage. Pockets of pus trapped beneath slough must be sought and liberated by debridement. Warm saline dressings (changed several times daily) are applied to infected areas. Cultures are taken and antibiotics chosen by sensitivity studies. Prolonged antibiotic therapy is not necessary if drainage and dressings are adequate. Skin grafts will not survive

a virulent, invasive infection, but grafting should be done as soon as the infection is under control.

General Supportive Measures.

Chronic infection, exudative loss of protein, the catabolic response to stress, and the anorexia and depression caused by pain and toxemia can produce rapid nutritional depletion in the severely burned patient. The anemia that is often present is caused by hemolysis at the time of burning and subsequent inhibition of erythropoiesis by infection. These changes must be prevented by administering a high-caloric, high-protein intake at the outset of therapy (see p. 95) and giving vitamin supplements and blood transfusions to keep the Hgb. above 12 Gm.%.

OTHER EMERGENCIES

HEAT STROKE
(Sunstroke)

Heat stroke may be rapidly fatal. It is characterized by fainting, fever, and cessation of sweating due to the failure of the hypothalamic heat-regulating mechanism during exposure to high temperatures. Absence of sweating is an important premonitory sign. Prodromal symptoms include headache, dizziness, nausea, and visual disturbances. Convulsions sometimes occur. The skin is hot, flushed, and dry. The pulse is rapid and full   Rectal temperatures may be as high as 108-112°F. (42-44°C.). Hydration and salt content of the body are normal.

The objective of treatment is to reduce high body temperature by means of ice packs, sponging with cold alcohol, immersion in a cold bath (10°C.), and/or ice water enemas. Massage the skin to maintain peripheral vasodilatation while cooling. Reduce rectal temperature promptly to 102°F. (39°C.). Proceed more slowly thereafter.

HEAT CRAMPS

Heat cramps are painful spasms of the voluntary muscles of the abdomen and extremities due to depletion of body salt by sweating during prolonged exposure to heat. Heavy manual labor in a hot environment may result in the loss of 3-4 L. of sweat (containing 0.2-0.5% sodium chloride) per hour. In addition to cramps, there may be muscle twitchings. The skin is moist and cool, and body temperature is normal or only slightly elevated. Laboratory studies reveal a low serum sodium. Persons working in high temperatures should take an increased amount of salt, either (1) by adding 1 level tsp. of table salt to each quart of water; or (2) by taking one 1 Gm. (15 gr.) sodium chloride tablet with every 1-2 glasses of water.

Remove the victim to a cool place. Cramps will frequently subside on rest alone. Give sodium chloride, 1 Gm. (15 gr.) by mouth

every hour with 1-2 glasses of water until 15 doses have been administered. In severe cases, give 1000-2000 ml. of physiologic saline I.V.

COLD INJURY

The fundamental pathologic process in all types of cold injury is the same. Exposure to cold causes arteriolar and capillary spasm, and ischemia and tissue hypoxia produce varying degrees of tissue damage. Because the endothelium of capillaries is damaged, blistering and edema due to plasma leakage are characteristic of severe cold injury. The most serious destruction occurs in the skin and subcutaneous tissues, since cooling is greatest in these superficial structures

In the great majority of cold injuries the tissues are not actually frozen but suffer from ischemia and thrombosis in the smaller vessels induced by wet cold at temperatures above freezing (e.g , trench foot). True freezing injury (frostbite) does not occur until the skin temperature drops to 32° F (0° C.)

The likelihood of cold injury is increased by immobility, venous stasis, occlusive arterial vascular disease, and previous cold injury.

Prophylaxis. - "Keep warm, keep dry, and keep moving "
Wear sufficient windproof and water-repellant clothing, change wet garments and footgear as quickly as possible, and maintain the circulation by frequently exercising the arms, legs, fingers, and toes
Avoid constrictive clothing and shoes and prolonged dependency of feet.

Chilblain (Pernio).

Chilblain is a relatively mild and superficial form of cold injury. It is most common on the dorsum of the hands of outdoor workers. It may also affect the legs, particularly the anterior tibial surface in young women. The acute form is characterized by a transient bluish-red appearance of the skin and mild edema, often associated with itching or burning which is aggravated by warmth. Repeated exposure may cause chronic manifestations: increased swelling, reddish-purple discoloration of the skin, blisters, and hemorrhagic ulcers which heal slowly and leave pigmented scars

Treatment consists of rewarming the affected area promptly to body temperature. Avoid trauma, rubbing, massage, and excessive heat. Elevate the part if edema is present. Protect from infection by gentle cleansing and aseptic dry dressings over open vesicles and ulcers

Trench Foot (Immersion Foot) and Frostbite.

Trench foot (which may also affect the hands) results from prolonged exposure to cold at temperatures from just above freezing to 50° F. (10° C.), often in a damp environment and usually in connection with immobilization and dependency of the extremities. Immersion foot is caused by exposure to water at temperatures below 50° F. (10° C.), usually for more than 12 hours. The first symptom is usually an uncomfortable coldness, followed by numbness. Throbbing, aching, or burning and varying degrees of redness, cyanosis,

edema, blistering, and skin necrosis occur after rewarming   In
severe injuries the tissues become black, dry, and mummified,
and later develop a sharp line of demarcation.

Frostbite is due to actual freezing of the tissue fluids after ex-
posure to freezing temperatures. The exposure necessary to pro-
duce frostbite varies from a few seconds to several hours, depend-
ing upon the temperature of the environment

Local sensations of coldness or stinging pain give way to numb-
ness   The skin becomes pale after severe exposure. A phase of
hyperemia may occur after rewarming during which the skin is
acutely painful, warm, and red or bluish in color   These changes
are followed by varying degrees of edema, blistering, and skin
necrosis, depending upon the depth of injury. The phase of hyper-
emia and local warmth gives way to cyanosis, hyperhidrosis, and
coldness, and persistent, burning pain often sets in. Pre-necrotic
skin becomes dark brown and finally black   These areas of dry
gangrene eventually mummify; 1-2 months are required for separ-
ation and epithelization

A Immediate Treatment:
   1. Local measures –
      a. Rewarm rapidly by immersion in water at 90-104° F
         (32-40° C.), with body heat, or by exposure to warm air
         Do not expose to an open fire   Maintain general body
         warmth
      b. Avoid trauma – All casualties with foot involvement are
         litter cases   Do not rub or massage the affected part.
      c Prevent infection – Cleanse the part gently with bland or
         hexachlorophene soap   Dressings are not necessary if
         the skin is intact
   2 Administer tetanus toxoid or antitoxin (see p. 130).

B General Treatment·
   1 Local measures –
      a. Absolute bed rest is required when the feet are involved
         Protect pressure sites (e g , heels) by the use of pillows.
      b. Slight elevation of the part controls edema   Marked ele-
         vation diminishes blood flow and is contraindicated
      c Expose closed lesions to room air at a temperature of
         70-74° F. (21-23° C.).
      d Rigid asepsis is mandatory in dressing open lesions   Use
         sterile dry gauze dressings, loosely applied
      e. Debride conservatively and advise amputation only when
         demarcation is definite   Remove superficial necrotic
         tissue with aseptic precautions, and cover large open
         areas with skin grafts as early as possible. In general,
         the tissue loss in cold injury will be less than appeared
         likely at the outset.
   2. Antibiotics – Treat invasive infection with specific antibiotics
      chosen by sensitivity studies on cultures of exudate   If bac-
      teriologic facilities are not available, use penicillin empiri-
      cally (see p 614).
   3 Anticoagulants, sympathetic block, sympathectomy, gangli-
      onic blocking agents, and prohibition of smoking are of no
      proved value in the limitation of tissue loss due to cold injury
C. Treatment of Sequelae:
   1. Neurologic measures – Late sequelae such as hyperhidrosis,

coldness, cyanosis, edema, chronic ulcers, and pain may be palliated by sympathectomy.

2. Orthopedic measures - Foot pain on weight-bearing should be treated with well-fitted shoes and the use of pads, supports, and other orthopedic devices. Contractures due to fibrosis of muscles pose special orthopedic problems.

ELECTRIC SHOCK

Electric shock produces 4 significant types of reaction, 1 or all of which may occur depending upon the circumstances: (1) Electric burn at the point of contact with the current and in the tissues through which heavy current passes. The skin and adjacent structures are intensely charred to variable depths. (2) Loss of consciousness, which may be momentary or prolonged. With recovery there may be muscle pain, fatigue, headache, and nervous irritability. (3) Respiratory paralysis, which is accompanied by coma and may persist for 6-8 hours if artificial respiration is provided (4) Ventricular fibrillation. The patient is comatose and pulseless, and no heart sounds can be heard. Respirations cease, and death supervenes within a few minutes unless circulation is restored by cardiac massage (see p. 39).

Alternating current is more dangerous than direct current. With alternating currents of 25-300 cycles, low voltages (below 200) tend to produce ventricular fibrillation; high voltages (over 1000), respiratory failure; intermediate voltages (220-1000), both

Treatment.

Emergency measures consist of (1) immediate removal of the patient from the electric current; (2) administration of artificial respiration (see p. 6); and (3) treatment of any cardiac arrhythmia (see pp. 37 and 39). Electric burns are usually third degree and require skin grafting after sufficient time has elapsed for demarcation.

DROWNING

Respiratory obstruction is the primary disturbance in drowning. Spasm of the larynx usually develops, followed by acute oxygen deprivation and respiratory arrest; only a small amount of water may be aspirated. The stomach, however, may be filled with water. The body is cold and the face cyanotic and congested.

Emergency Treatment.

A. Clear the airway. Remove mucus and foreign matter manually from the nose and throat and drain water from the respiratory tract by gravity. Use suction if available.

B. Loosen or remove constricting clothing.

C. Begin mouth-to-mouth artificial respiration immediately (see p. 6) and continue until spontaneous respiration returns or until heart action is stopped and death absolutely certain.

D. Keep the patient comfortably warm.

CARBON MONOXIDE POISONING

Carbon monoxide is produced by the incomplete combustion of carbon-containing material; it can therefore be emitted by any flame or combustion device, including gas stoves, coal-burning heaters, and automobiles. Carbon monoxide combines with hemoglobin to form the stable compound, carboxyhemoglobin, which is incapable of carrying oxygen. Tissue hypoxia results. Manifestations include headache, faintness, dizziness, tinnitus, vomiting, cherry-red skin and mucous membranes, loss of memory, collapse, and coma. When the patient's blood is boiled or shaken with 1-2 volumes of sodium hydroxide, normal blood becomes black or brown-black but blood containing high levels of carboxyhemoglobin remains red.

Emergency treatment consists of removing the patient to fresh air and keeping him warm and at rest. Give 100% oxygen by mask for 1 hour. If respirations are depressed, positive pressure breathing of 100% oxygen will reduce the blood carboxyhemoglobin below the dangerous level. If BP is markedly depressed, use a vasopressor drug (see p. 623).

SNAKE BITES

The venom of poisonous snakes may be neurotoxic or hemotoxic. Bites from poisonous snakes are characterized by the appearance of 2 puncture wounds; nonpoisonous snakes leave semicircular rows of tooth marks. The manifestations of poisonous snake bite are local pain, redness, and swelling followed rapidly by nausea, vomiting, and collapse. Hemotoxic venoms cause hemolysis, local hemorrhage, and bleeding from the mucous membranes. Neurotoxins tend to cause respiratory failure.

Keep the patient recumbent and quiet. Apply a tourniquet proximal to the bite and make cross-incisions deeply through the bite; apply suction by mouth or by means of a suction cup, breast pump, or other device. Place an ice pack on the wound.

Treat respiratory failure with artificial respiration (see p. 6) or positive pressure breathing with oxygen (see p. 84). Give specific antivenin as soon as possible. General supportive measures such as transfusions, sedation, and corticosteroids should be given as indicated. Marked local tissue necrosis may occur, and may require grafting.

BLACK WIDOW SPIDER BITE

The venom of the black widow spider is quite toxic. Local reactions consist of pain, redness, and swelling. Systemic symptoms may include generalized muscular pains, abdominal cramps, nausea and vomiting, and collapse. The abdominal muscles may become rigid, in which case the clinical picture resembles that of an acute abdominal emergency. However, the patient with black widow spider bite is extremely restless.

Treatment of the local wound is apparently not helpful. Administer Antivenin (Latrodectus mactans), N.N.D., when the systemic reaction is severe. For convulsions or muscle cramps, give

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calcium gluconate, 10 ml. of 10% solution, I.V., and repeat p.r.n
Additional measures consist of hot baths and control of restlessness
with barbiturates (see p. 607).

ACUTE RADIATION INJURY

Ionizing radiation injury may be produced by alpha particles,
beta particles, gamma rays, and neutrons   Alpha and beta particles
have low penetrating ability and are only important when a substance
emitting these particles contaminates the body surface or is inges-
ted.   Gamma rays and neutrons have great penetrating power and
are primarily responsible for acute radiation injury in nuclear ex-
plosions or accidents.

Whole body radiation of less than 200 r is not lethal. With 300 r,
about 20% deaths can be expected. The death rate approaches 100%
with 600 r, which is the exposure of an unprotected person about
three-quarters of a mile from the air-burst of an atomic bomb   In
practice, the extent of radiation exposure in each individual will not
be known, and treatment must be symptomatic and expectant.

Clinical Features.
  A. Sublethal Radiation Exposure (100-200 r)· Nausea and vomiting
     usually occur within the first 24 hours and subside spontaneous-
     ly.   There may be no late symptoms   The delayed development
     of leukopenia may be the only evidence of radiation damage
  B. Potentially Lethal Radiation Exposure (300-450 r)· Nausea and
     vomiting will occur in all cases, usually within a few hours,
     but will subside within about 24 hours   After an asymptomatic
     period of 1-3 weeks there may be oral and cutaneous lesions,
     severe infections, purpura, epilation, bloody diarrhea, leuko-
     penia, and anemia
  C. Lethal Radiation Exposure (Over 600 r): Persistent vomiting
     develops within 2 hours after exposure and is followed by fever,
     diarrhea, severe dehydration, extreme prostration, and death
     in a few hours or days

Treatment.
  No specific measures are available
  A. Decontamination: Monitoring devices are valuable in determin-
     ing the amount of radioactivity of the skin, clothing, food, and
     water. Surface and subsurface explosions are most likely to
     produce heavy contamination of the environment. Dispose of
     all contaminated clothing as soon as possible and before enter-
     ing a noncontaminated area. Cleanse all exposed skin with soap
     and water or a detergent   Wipe the skin with any noncontami-
     nated cloth, paper, etc.
  B. Symptomatic Treatment: Use sedatives (see p. 607), narcotics
     (see p. 605), and antinauseant drugs (see p. 622) as required.
     Combat dehydration and electrolyte imbalance in the usual man-
     ner (see p. 99), using the oral route if possible. Parenteral
     fluids are not likely to be in sufficient supply in case of mass
     casualties. Antibiotics, corticosteroids, and whole blood and
     plasma should be reserved for specific indications and not used
     indiscriminately in radiation injury.

EYE INJURIES

In severe eye injuries it is important for the nonspecialist to avoid causing further damage by unnecessary manipulation and to refer the patient immediately to an ophthalmologist. Local anesthetics, dyes, or other medication placed in an eye with a damaged corneal epithelium **must be sterile** Examinations should be made with a well-focussed light and with a magnifying glass or loupe

Foreign Bodies.

The most common eye injury is caused by a flying particle which lodges in the conjunctiva or cornea. The history may warrant taking an x-ray of the eye, since an intraocular metallic fragment may cause minimal early symptoms. If a foreign body has perforated or deeply penetrated the cornea or globe, the patient should be referred at once to a specialist; only superficial foreign bodies should be removed in the office or emergency room.

A Conjunctival Foreign Bodies:
1. Evert the lid, if necessary by pressing gently downward across its outer surface with a match or applicator stick while pulling upward on the eyelashes
2. Remove free particles with a moist cotton-tipped applicator or by gentle irrigation with warm sterile saline solution

B. Corneal Foreign Bodies:
1. Instill 2 drops of sterile 0.5% tetracaine (Pontocaine®) solution into the eye as a topical anesthetic.
2. Superficial particles may be removed with a moist cotton-tipped applicator.
3 Imbedded particles are removed with a delicate instrument such as the point of a hypodermic needle, a Bard-Parker blade, or a special "hockey stick" spud. A dental drill of the burr type is often useful for removing rust rings.
4 After-care of imbedded particles –
   a. If the particle has been present over 24 hours, instill 2 drops of sterile 5% homatropine hydrobromide solution into the conjunctival sac to dilate the pupil.
   b. Instill an antibiotic ophthalmic ointment, such as polymyxin B–bacitracin (Polysporin®) or neomycin–polymyxin B–bacitracin (Neosporin®), t.i.d.
   c. Control pain with meperidine (see p. 606) or codeine (see p. 606) rather than with the repeated use of a topical anesthetic.
   d. Apply a soft pad over the eye and bandage or tape it in place under moderate pressure. Keep the injured eye patched and instruct the patient not to use his eyes until the corneal surface has healed. Staining of unhealed areas of epithelium with sterile 2% fluorescein may be helpful in follow-up evaluation of healing.
   e. Examine the eye daily for signs of infection such as a red eye with a cloudy cornea, purulent discharge, or significant photophobia. If any of these signs appear, refer the patient immediately to an ophthalmologist.

Corneal Abrasions.

These are usually superficial and minor but if inadequately treated may lead to recurrent erosion and pain. Instill 2 drops of

sterile 2% fluorescein solution as a stain to indicate the extent of
the abrasion. If the abrasion is extensive, dilate the pupil with 2
drops of sterile 5% homatropine solution. Instill an antibiotic oint-
ment such as polymyxin B-bacitracin (Polysporin®) or neomycin-
polymyxin B-bacitracin (Neosporin®) t.i.d. Dress and follow as
for corneal foreign body.

Burns of the Eye.
  A. Thermal: These are usually associated with face or body burns.
     If the conjunctivas are involved, adhesions may develop. If the
     cornea is severely damaged, perforation or infection is apt to
     occur and an ophthalmologist should be consulted. Dilate the
     pupil with sterile 5% homatropine solution, instill an antibiotic
     ointment t.i.d., relieve pain with cold compresses and sys-
     temic analgesics or narcotics, and keep the eyes closed.
  B. Ultraviolet: Arc welding or bright sunlight may produce a
     superficial painful keratitis which usually heals without com-
     plications within 24-48 hours. Relieve pain by an initial in-
     stillation of sterile 0.5% tetracaine (Pontocaine®) solution and
     subsequently with systemic analgesics or narcotics. Keep the
     eyes bandaged and apply cold compresses until the initial re-
     action subsides.
  C. Chemical: Flush out the conjunctival sac immediately with tap
     water for several minutes. Neutralizing solutions are unnec-
     essary and may even be harmful. If fluorescein stain after
     irrigation indicates that corneal damage has occurred, treat as
     for thermal burn and refer to an ophthalmologist for definitive
     care.

Contusions of the Eyeball.
     If ophthalmoscopic examination shows any evidence of internal
hemorrhage or damage to the iris, lens, or retina, specialist care
is indicated. Any injury severe enough to produce intraocular hem-
orrhage may cause secondary hemorrhage and intractable glaucoma.
Such patients should be put at absolute bed rest for 1 week with both
eyes bandaged in order to prevent further bleeding.

Lacerations of the Eyeball.
     Minor lacerations of the conjunctivas or globe without prolapse
of intraocular contents may be treated by fine silk suturing More
extensive lacerations of the globe with prolapse or leakage of intra-
ocular contents require specialist care since they may lead to
blindness or loss of the globe itself. Emergency and temporizing
measures include dilatation of the pupil with sterile 5% homatropine
solution, instillation of an ophthalmic antibiotic ointment t.i.d.,
systemic antibiotics (see p. 614), analgesics or narcotics, and bi-
lateral eye bandages. If the wound is extensive and loss of contents
has been great enough to preclude function, enucleation may be indi-
cated. If uveal tissue has been injured and the eye is retained,
sympathetic ophthalmia may occur 2 weeks to several years after
the initial injury. Fortunately, this dread complication is rare.

COMMON SOFT TISSUE INJURIES

Contusions.

Nonpenetrating blunt trauma produces tissue damage and interstitial hemorrhage, but the superficial soft tissue injury itself is rarely significant. Underlying fracture or damage to an internal organ must be ruled out

Contusions usually require no treatment. The application of an ice pack within the first few hours may limit interstitial hemorrhage and subsequent ecchymosis.

Hematomas.

A large subcutaneous or intramuscular hematoma occasionally results from contusion. If subsidence is slow, aspiration with a No. 15 or 17 gauge needle may be attempted under local anesthesia but this is usually unsuccessful. Before withdrawing the needle, inject 1500 U.S.P. units of hyaluronidase (Wydase®) and repeat the injection daily or oftener. Incision and evacuation of a hematoma is rarely necessary. Aseptic technic is important when aspirating or evacuating hematomas since they provide ideal conditions for bacterial growth

Lacerations

The basic steps in management are debridement, irrigation, and wound closure, usually under local procaine anesthesia. Always examine for damage to nerves and tendons and for the presence of foreign material. Lacerations of the hand (see p. 463) and those associated with loss of skin (see p. 439) require specialist care.

A. Primary vs. Delayed Wound Closure: Repair of a laceration should be done as soon after injury as possible. The more heavily contused or contaminated the wound, the more important it becomes to treat within a few hours. Certain wounds, however, may occasionally be safely sutured as long as 24 or more hours after injury. This applies to neatly incised lacerations occurring under relatively clean conditions, especially around the head and neck where blood supply is generous and a good cosmetic result is important. Catgut is used in the deeper layers; and fine, nonabsorbable sutures are used in the skin. Heavily contaminated wounds or those considered too old for primary closure should be covered with a single layer of petrolatum gauze and packed lightly open with fluffed gauze; 48-72 hours later the wound should be inspected and loosely closed with sutures or flamed adhesive strips if it appears clean.

B. Antibiotics: Most small or clean lacerations do not require antibiotic therapy. When contamination is marked or when primary closure must be done late, systemic penicillin therapy is justified (see p. 614).

C. Tetanus Prophylaxis: (See p. 130.) This is indicated if soil contamination is suspected.

Penetrating Wounds.

The chief complications of penetrating wounds are (1) perforation of a viscus, (2) introduction of pyogenic or tetanus organisms, and (3) retained foreign body. Treatment must be individualized.

Clean puncture wounds and through-and-through missile wounds which cause no serious damage may require only observation. Penetrating injuries which drive clothing or other foreign material into the wound usually require exploration and debridement. Tetanus prophylaxis is often indicated (see p 130).

Foreign Bodies.
Any open wound may contain a foreign body Attempt removal of a foreign body only after adequate localization by (1) x-rays in 2 or more planes with lead markers on the skin, (2) placing identifying marks on the skin under fluoroscopy, or (3) inserting a needle down to the foreign body under fluoroscopy Small (less than 1 cm), deep, inert foreign bodies can usually be left alone and observed by x-ray several months later A foreign body should be removed if it protrudes through the skin, if it causes pain, if it consists of or is contaminated with dirt, cloth, wood, or other material likely to cause infection or reaction; if the wound is infected or draining; or if it may migrate to or impinge upon important structures Tetanus prophylaxis should be given as indicated (see p 130).

HUMAN BITE

Deep penetrating wounds of the knuckles are common, with involvement of the metacarpophalangeal joint and extensor tendon When the hand is opened, the proximal glide of the extensor tendon carries infecting organisms proximally into anaerobic sites on the back of the hand. Cellulitis usually appears within 24-72 hours and may extend rapidly. Necrosis, abscess formation, and marked pain and swelling are the typical findings

Emergency treatment consists of irrigation and debridement, extending the wound as required. Aerobic and anaerobic cultures should be taken and tests made for antibiotic sensitivity. Do not suture the wound, but cover it with petrolatum gauze and a dry dressing and splint the hand and wrist as necessary. Observe for infection, and administer full doses of penicillin (see p. 614) pending the results of antibiotic sensitivity tests.

If such a wound is seen late, obtain cultures, drain abscesses, place the patient at bed rest with the part elevated, apply hot moist packs, and give penicillin until the antibiotic of choice can be chosen on the basis of sensitivity studies

ANIMAL BITES

Dog Bites. (See also Rabies, below.)
Penetrating small wounds should be thoroughly irrigated and left open. Lacerations are debrided, irrigated, and sutured according to the usual principles (see p. 31). Tetanus prophylaxis is given as indicated (see p. 130).

Cat Bites.
Cat bites are more likely to cause infection than dog bites. Cleanse the area thoroughly with soap and water, apply a dry sterile

dressing, splint the part if possible, and observe for infection. If cellulitis occurs, administer penicillin (see p. 614) pending anti-biotic sensitivity studies. Tetanus prophylaxis is given as indicated (see p. 130).

RABIES*

Rabies is a viral encephalitis transmitted through the saliva of an infected animal. Humans are usually inoculated by the bite of a rabid dog. Since the established disease is invariably fatal, early preventive treatment is of paramount importance.

The incubation period varies in humans from 10 days to sev-eral months The onset is with pain and numbness around the site of the wound followed by fever, irritability, malaise, and spasms of the muscles of swallowing. Paralysis and convulsions occur terminally. Absolute quiet and anticonvulsant drug therapy are indicated for established cases

Prophylaxis.

Do not destroy the animal, but confine it under veterinary ob-servation for 1 week, if it becomes rabid, the animal should be sacrificed and its brain cells examined for Negri bodies. Consult the local health authorities to determine if any cases of rabies have been reported recently.
A. If the animal is not overtly rabid and has been impounded -
1. Cleanse the wound carefully with bland soap and water and irrigate copiously with saline solution.
2. Debride devitalized tissues, and repair as indicated,
3 Give tetanus prophylaxis as indicated (see p. 130).
4 Antirabies immunizations are advised only if the animal becomes rabid
B. If the animal is known to be rabid, or if the attack was unpro-voked and the animal has been killed or has escaped -
1. Cauterize the wound with fuming nitric acid if it is possible to do so within 24 hours
2. Irrigate copiously with saline and cleanse with soap and water
3. Antirabies immunization is indicated if the animal is known to be or strongly suspected of being rabid.
C. Antirabies Immunization.
1. Rabies antiserum (hyperimmune serum) (horse) - Horse serum confers a transient passive immunity and should be given within 72 hours of the bite if the incubation period is likely to be short, as in facial bites and extensive bites of the upper extremities. The usual horse serum sensitivity precautions must be taken. In addition to antiserum, rabies vaccine must also be used to produce active immunization.
2. Rabies Vaccine (Duck Embryo), N.N.D., 1 ml. subcut. daily for 14 days. Rabies vaccine occasionally causes seri-ous encephalitis, but duck embryo vaccine is reported to cause fewer neurologic sequelae than older preparations.

*Physicians in rabies endemic areas are advised to consult WHO Technical Report Series No. 121, 1957, prepared by the Expert Committee on Rabies of the World Health Organization.

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Preoperative and Postoperative Care

GENERAL CONSIDERATIONS IN PREOPERATIVE MANAGEMENT

PREOPERATIVE EVALUATION

The basic work-up for major surgery should include a complete history and physical examination, urinalysis, complete blood count (CBC), serology, chest x-ray, and, in patients over 50, an Ecg. and stool test for occult blood. For the older age group, a BUN and postprandial blood sugar are often advisable as screening measures. When blood transfusion is anticipated, blood typing and cross-matching should be ordered. Open wounds and infections usually require bacteriologic investigation. In addition to the foregoing more or less routine studies, all significant complaints and physical findings should be adequately evaluated by appropriate special tests, examinations, and consultations

OPERATIVE PERMIT; CONSULTATIONS

The patient or his legal guardian must authorize surgery by signing a permit which specifically names the procedure which is planned. The nature, risk, and probable result of this procedure should be made clear to the patient and/or a responsible relative or guardian preoperatively. Therapeutic abortions and operations which adversely affect the sexual or childbearing functions should be undertaken only with the concurrence in writing of the marital partner.

Emergency, life-saving operations may have to be done without a permit. If possible in these cases obtain adequate consultation and inform the director of the hospital in advance.

The opinion of a qualified consultant should be obtained whenever it may be of benefit to the patient or whenever it may be of medicolegal importance.

NONSURGICAL DISORDERS AFFECTING OPERATIVE RISK

HEART DISEASE

Conditions Which Increase Operative Risk.
Past or present involvement with any of the following definitely increases the hazard of surgery:
- A. Congestive failure or limited myocardial reserve, as a result of rheumatic or arteriosclerotic heart disease.
- B. Myocardial infarction or angina pectoris.
- C. Serious arrhythmia (fibrillation, flutter, or block).
- D. Hypertension associated with chronic nephritis. Moderate hypertension alone does not affect the prognosis significantly unless renal or cardiac complications are present.
- E. Aortic stenosis (rheumatic or arteriosclerotic).
- F. Syphilitic cardiovascular disease.
- G. Congenital heart disease. The risk is primarily related to the functional state of the heart (see p. 234).

Special Diagnostic Measures.
In addition to the studies included in the routine initial workup (see p. 34), the following are useful in the evaluation of the cardiac patient.
- A. Electrocardiography· The Ecg. is of little help in determining operative risk, but it is of diagnostic value and useful as a baseline for evaluating subsequent changes in the myocardium and conduction system
- B. Venous Pressure Determination: (Normal range = 6-10 cm. of water.) Venous pressure should be determined when serious heart disease is present and particularly when congestive failure is suspected.

    Technic of direct method: Keep the patient at rest for 15 minutes. The site of venipuncture should be kept at the level of the junction of the superior vena cava and the right atrium throughout the procedure. If the patient is sitting, this is at the level of the third sternocostochondral junction. If the patient is supine, support the arm so that the vein will be on a horizontal plane below the base of the xiphoid at a distance equal to one-third of the anteroposterior diameter of the chest.

    Insert a large needle (No. 18) into the antecubital or other convenient arm vein. Using a three-way stopcock, attach to the needle a calibrated glass spinal manometer and a syringe containing saline or 2% sodium citrate, which is used to fill · the whole system. The entire apparatus must be sterile. Venous pressure is read at the point at which the fluid level stabilizes in the upright manometer.

    More simply, the venous pressure is determined by measuring the height above the right atrium at which the fluid level in an intravenous tubing comes to rest when the bottle is temporarily disconnected.
- C. Circulation Time: Arm-to-tongue time is normally 10-16 seconds as determined with Sodium Dehydrocholate, N.N.D. (Decholin®). Other methods have different values. Circulation

time should be determined in serious heart disease even in the absence of physical signs. Prolonged values indicate early left heart failure.

Technic for circulation time determination: Through an antecubital vein, rapidly inject 5 ml. of a 20% solution of Sodium Dehydrocholate, N.N.D. (Decholin®). Note the time between injection and the moment when the patient first notices a bitter taste. Prior to the test the patient must be instructed to give a specific signal immediately when this occurs. The test may be repeated without withdrawing the needle. Extravasation is very painful and must be avoided.

D. Pulmonary Function Tests: These tests should be done whenever dyspnea on exertion is significant and in all patients who are to undergo thoracic surgery (see p. 196).

E Renal Function Tests: Patients with hypertensive and arteriosclerotic heart disease should have BUN, creatinine, and PSP excretion tests. An intravenous urogram is also frequently indicated if organic urinary tract disease is suspected (see p. 373).

F. Other Tests: Phonocardiography, angiocardiography, cardiac catheterization, and other highly specialized examinations are rarely required except in congenital heart disease.

Preoperative Preparation.

Prior to surgery the cardiac patient should be in the best cardiac status possible, with special attention directed toward correction of electrolyte imbalance and anemia If possible, delay surgery for at least 3 weeks after correction of congestive failure and 3-6 months after myocardial infarction.

Principles of Care During and After Operation.

Avoid hypotension, hypoxia, excessive sodium, fluid, and blood administration; and undue pain or excitement in the cardiac patient. These stresses may precipitate failure, myocardial infarction, or serious arrhythmia, which are the major cardiac complications

Treatment of Cardiac Complications.

A. Treatment of Acute Pulmonary Edema (Acute Congestive Failure): Acute pulmonary edema is a grave emergency. **Act immediately.** It is most likely to occur postoperatively and is often precipitated by stress, transfusion of too much blood, or excess administration of sodium-containing fluids intravenously. Myocardial infarction or an attack of atrial fibrillation with a rapid ventricular rate may be the precipitating factor.

1. Position – It is essential to place the patient in a sitting position in bed or in a chair in order to decrease venous return to the heart.

2. Morphine Sulfate, U.S.P., 15-30 mg. ($\frac{1}{4}$-$\frac{1}{2}$ gr.) I.V. or I.M., depresses pulmonary reflexes, relieves anxiety, and induces sleep.

3. Give oxygen in high concentrations, preferably by mask.

4. Reduction of blood volume is accomplished in 1 of 2 ways:

    a. By application of rubber tourniquets or BP cuffs to 3
       extremities with sufficient pressure to obstruct venous
       but not arterial flow. Rotate the tourniquets every
       10-15 minutes by placing a tourniquet on the fourth ex-
       tremity and releasing 1 of the others.
    b. By venesection of as much as 1 L. of blood in successive
       bleedings of 500, 300, and 200 ml.

  5. Rapid digitalization - When a patient is already receiving
    digitalis the problem is more difficult. Once it has been
    determined that the patient is only partially digitalized,
    give Cedilanid-D® cautiously I.V. in an initial dose of 0.4
    mg. ($1/150$ gr.) followed by 0 2 mg. ($1/300$ gr.) at half-
    hourly intervals until there is improvement or evidence of
    digitalis toxicity.

  6. Aminophylline, U.S.P., 0.25-0.5 Gm. ($4-7^1/2$ gr.) slowly
    I.V., may be helpful   Rectal suppositories may also be
    used.

B. Treatment of Congestive Failure· Symptoms and signs include
  dyspnea, orthopnea, rales at lung bases, venous and hepatic
  engorgement, dependent edema, prolonged circulation time,
  and increased venous pressure.

  1. Eliminate or control precipitating factors such as stress,
    sepsis, anemia, excess sodium administration, arrhythmias,
    and thyrotoxicosis.

  2. Rest in bed or chair with appropriate sedation decreases the
    work required of the heart.

  3. The diet should be bland, low-residue, and low in sodium
    (less than 0.6 Gm. of sodium or 1.5 Gm. of sodium chloride).
    With sodium restriction, fluids may be allowed ad lib

  4. Digitalis (see above)

  5. Diuretics (see p. 609)

  6. The patient will lose weight and dyspnea will subside as im-
    provement occurs. Activity can then be resumed gradually
    within appropriate limits.

C. Treatment of Acute Myocardial Infarction: Prolonged, usually
  severe precordial or substernal pain, sometimes radiating to
  the neck or upper extremities, is typical, but these symptoms
  may be masked by anesthesia or narcotics. Infarction is often
  associated with shock, congestive failure, and arrhythmias.
  Other manifestations include fever, leukocytosis, elevation of
  sedimentation rate and serum transaminase level, and charac-
  teristic Ecg. changes (which may be delayed).

  1. Rest - Complete bed rest for 2-4 weeks is advocated, with
    gradual resumption of activity as tolerated. Use sedatives
    as required.

  2. Relief of pain - Give Morphine Sulfate, U.S.P., 10-15 mg.
    ($1/6-1/4$ gr.) slowly I.V., or choose an alternative narcotic.

  3. Oxygen therapy is often necessary.

  4. Shock - If cardiac shock is present, treat with vasopressor
    drugs and rapid digitalization.

  5. Digitalis and drugs to control arrhythmia are prescribed for
    specific indications.

D. Treatment of Cardiac Arrhythmias: The treatment of the major
  arrhythmias is a complex problem calling for the close collab-
  oration of the internist and surgeon. However, the surgeon may

be required to give emergency treatment or initiate treatment until the services of an internist can be obtained.

1. **Atrial fibrillation** - Rapid atrial fibrillation decreases the efficiency of the heart and may lead to congestive failure. Emergency treatment consists of slowing the rate by adequate digitalization.

2. **Atrial flutter** - Rapid atrial flutter may lead to congestive failure. Digitalization will slow the rate, either by increasing the degree of block or by converting the flutter to sinus rhythm or atrial fibrillation. The final conversion is best supervised by one familiar with the treatment of this condition.

3. **Atrial paroxysmal tachycardia** - If vagus stimulation (carotid sinus pressure, gagging, Valsalva's maneuver) does not cause conversion, digitalization should be tried; if digitalization fails, quinidine or procainamide (Pronestyl®) must be used as for ventricular tachycardia (see below).

4. **Nodal paroxysmal tachycardia** - Treat as for atrial tachycardia.

5. **Ventricular paroxysmal tachycardia** - This arrhythmia is usually associated with severe myocardial damage, especially myocardial infarction. Quinidine is tried first.

   a. Quinidine -
      (1) Oral - Quinidine Sulfate, U.S.P., 0.4 Gm. (6 gr.) orally every 2 hours for 3 doses, if the attack is well tolerated and the patient is not in shock (otherwise, give parenterally). If the attack continues and quinidine toxicity does not appear, increase the dose to 0.6 Gm. (9 gr.) orally every 2 hours for 3 doses. This usually terminates the attack. If it does not, give the drug I.M. or use procainamide.
      (2) I.M. - Quinidine Gluconate, N.F., 0.8 Gm. (12 gr.), or 0.5 Gm. (7$\frac{1}{2}$ gr.) of quinidine base, may be given I.M. and repeated every 2 hours for 2-3 doses.

   b. Procainamide (Pronestyl®) - This drug causes hypotension; BP must be observed frequently during its use.
      (1) Oral - Procainamide Hydrochloride, U.S.P. (Pronestyl®), 0.5-1.5 Gm. (7$\frac{1}{2}$-22$\frac{1}{2}$ gr.) orally every 4-6 hours.
      (2) I.M. - 0.5-1 Gm. (7$\frac{1}{2}$-15 gr.) may be given I.M. and repeated in 4 hours.
      (3) I.V. - 1 Gm. (15 gr.) ampul in 10 ml. diluent given slowly at a rate of 50-100 mg. (3/4-1$\frac{1}{2}$ gr.) per minute to a total of 1 Gm. (15 gr.) with continuous BP determinations and, if possible, Ecg. control. Discontinue drug as soon as conversion takes place.

   c. Give a vasopressor drug for shock. Digitalis must be used cautiously in this disorder, and only in patients with cardiac failure in whom other drugs have failed to restore sinus rhythm.

6. **Ventricular fibrillation** produces cardiac arrest and requires cardiac massage (see below).

Prognosis.

Generalizations about the cardiac patient's prognosis for survival of surgery are difficult. Large series of cardiac patients have

shown an average mortality of only 3% after major abdominal and thoracic surgery. Patients with angina pectoris undergoing such procedures have a mortality risk of about 7-8%; most fatalities are from myocardial infarction. The danger is even greater (perhaps 10-15%) in patients who have had a previous myocardial infarct. As indicated above, serious arrhythmia and congestive failure, past or present, and hypertension with nephritis similarly affect prognosis in an adverse manner.

CARDIAC ARREST

Cardiac arrest consists of the unexpected cessation of the heartbeat secondary to acute myocardial hypoxia, toxicity, or conduction disturbance. Cardiac arrest occurs most frequently in the operating room during induction of anesthesia or in the course of surgery. Normal hearts may be so affected, but the presence of heart disease increases the susceptibility to arrest under adverse conditions. The general health, state of nutrition, blood volume, and pulmonary reserve of the patient, when subnormal, are contributing factors insofar as they predispose to hypoxia or hypotension during a surgical procedure

The commonest precipitating causes of cardiac arrest are as follows· (1) Shock, (2) hypoventilation, (3) airway obstruction, (4) overdosage of anesthetic, (5) drug effect or idiosyncrasy, (6) vago-vagal reflex, (7) manipulation of the heart, (8) hypothermia, and (9) myocardial infarction. Other conditions producing cardiac arrest include drowning, electrocution, asphyxia, poisoning with carbon monoxide and other gases, air embolism, and heart block.

Cardiac arrest occurs about once in every 750-1000 operations, one-third of which are minor procedures. The highest incidence is in operations on the lung or heart. Cardiac arrest occurs either as a result of ventricular standstill (asystole) or ventricular fibrillation. After cardiac arrest has occurred, it is impossible to distinguish ventricular standstill from ventricular fibrillation unless the heart is in view or an Ecg. is taken. However, the distinction is of no immediate importance.

Prophylaxis.
  A. Maintain adequate oxygenation at all times.
  B. Maintain adequate coronary blood flow.
  C. Prevent vagal effects. Preoperative administration of a vagal blocking agent (atropine or scopolamine) reduces the hazard of arrest. When bradycardia, A-V dissociation, or A-V nodal rhythm is produced during surgery by vagal stimulation, give atropine sulfate, 0.4-0.6 mg. ($1/150$-$1/100$ gr.) I.V. Avoid powerful traction on viscera, displacement of the heart, carotid sinus compression, etc.
  D. Prevent hypotension and treat immediately when it occurs.

Diagnosis.
  A. Premonitory Signs: The most constant signs of impending cardiac arrest are irregularity of rhythm, slowing of rate, and rapid fall of BP. Be alert for any or all of these premonitory changes during induction of anesthesia, intubation, or

extubation; when re-positioning or moving the patient under
anesthesia; after prolonged deep anesthesia, during periods of
hypoxia or hypotension; when manipulating the heart; and when
exciting vago-vagal reflexes (see above).

B. Pathognomonic Signs: **Verify cardiac arrest at once!** The
following signs are pathognomonic:

1. Absent pulse - Check for the pulse in a major vessel (e.g.,
carotid or femoral artery), or, when the abdomen is open,
the aorta.

2. Absent heart sounds - Listen to the precordium for the
heart beat. An ear to the chest is often the quickest method.
Do not wait for Ecg., BP apparatus, or special diagnostic
equipment.

NOTE· Absence of pulse and heart sounds means cardiac
arrest. **ACT IMMEDIATELY!**

Treatment.

Blood flow to the brain must be re-established within 4 minutes
if permanent cerebral damage or death is to be avoided.

A. Establish ventilation immediately. Start mouth-to-mouth
breathing until oxygen under positive pressure can be admin-
istered by mask or, preferably, endotracheal tube   Call for
an anesthetist.

B. Open the chest immediately. Speed is essential; enter the chest
in 30 seconds. Then send for surgical instruments, including
a rib-spreader.

1 A knife is the only necessary instrument.

2 No skin preparation or gloves are required

3. Make a left submammary incision through the fourth or fifth
interspace, spread the ribs wide enough so that the hand and
wrist can be inserted

C. Begin manual compression of the heart (cardiac massage) at
once

1. Compress the heart rhythmically 70-80 times/minute,
either against the sternum or between the fingers and palm
Open the pericardium longitudinally if response is not im-
mediate   Continue massage with 1 or both hands.

2 Establish coronary and brain circulation quickly. If manual
compression is effective, a distinct peripheral pulse is felt
The pulse should be monitored constantly by an assistant.
Continue manual systole until the myocardium is oxygenated
and pink, otherwise, strong contraction will not occur and
defibrillation is impossible. The pupils should remain con-
stricted if blood flow to the brain is adequate.

3. Be sure that the lungs are ventilating.

4. Intracardiac drugs - For the soft flabby heart (with or with-
out fibrillation), inject epinephrine into the right ventricle:
dilute 1 ml. of 1·1000 solution in 10 ml. saline; inject 2-5
ml.; and repeat p.r.n. If epinephrine is not available, in-
ject calcium gluconate, 3-5 ml. of 10% solution. No other
drugs are necessary.

D. Defibrillation: Ventricular fibrillation may be present at the
outset or may develop during manual compression. It is rarely
reversible except by electric shock. Proceed as follows:

1. First have an oxygenated (pink) myocardium with good tone.

It is futile and hazardous to attempt to defibrillate a cyanotic, flabby heart.

2. Pericardium must be open

3 Apply defibrillator poles on opposite sides of ventricles.

4 Set defibrillator for 0.1 second, 110-160 volts, 1.5 amperes, and deliver a shock

5. If fibrillation continues after the first shock, repeat quickly up to 5 or 6 times.

6. If defibrillation is not successful, continue massage, administer intracardiac epinephrine if indicated, and repeat shock when the condition of the myocardium is optimal

E. Persist in efforts to restore the heart beat as long as the myocardium maintains tone, color, and responsiveness Resuscitation may be successful after 6 hours of manual compression. Failures are usually due to delay in treatment, poor resuscitation technic, or inherent cardiac disease

F. Chest closure is routine Establish intercostal tube drainage and close with catgut. Give intensive postoperative antibiotic therapy
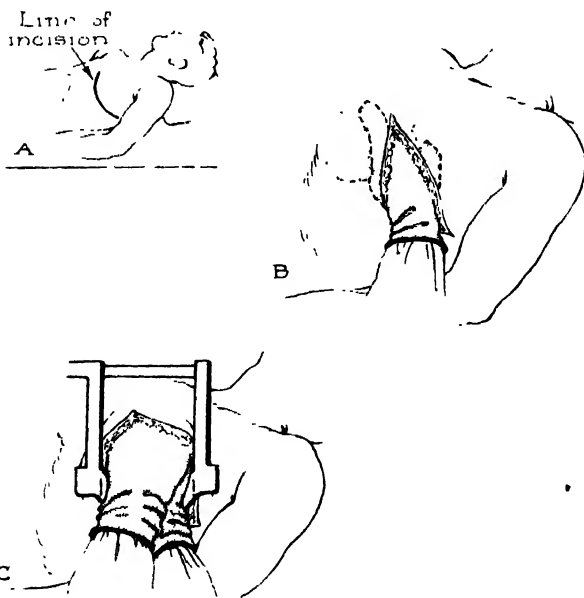
Cardiac Massage. (A) Position of patient for cardiac massage. (B) and (C) Insertion of 1 hand or 2 hands for rhythmic compression of heart.

Postoperative Care.

Cerebral edema caused by hypoxia is a factor in post-resuscitative death. Hypothermia may be a useful adjunct in the treatment of this condition. When there is evidence of cerebral damage, maintain the patient at 86-95°F. (30-35°C.) for about 72 hours postoperatively. This reduces cerebral edema and metabolism.

Prognosis.

Good results are proportional to the speed and skill of resuscitative efforts. When arrest occurs in the operating room, it is possible to save about 75% of the patients. The percentage of successful resuscitations following cardiac arrest in other areas of the hospital is increasing as education and facilities are provided. Ventricular fibrillation caused by acute myocardial infarction has occasionally been treated successfully by cardiac massage and defibrillation. It is the responsibility of every physician to acquire the judgment, skill, and decisiveness needed to accomplish cardiac resuscitation.

RESPIRATORY TRACT DISEASE

Operative morbidity and risk are affected adversely by acute or chronic respiratory tract diseases.

Acute Conditions.

Acute respiratory tract infection (coryza, pharyngitis, tonsillitis, laryngitis, bronchitis, or pneumonitis) is a contraindication to elective surgery because it is associated with an increased postoperative incidence of atelectasis and pneumonitis. If emergency operation must be undertaken in the presence of acute respiratory tract infection, avoid inhalation anesthesia if possible, employ prophylactic measures for atelectasis postoperatively (see p. 55), and administer antibiotics if the infection is marked or progressive. Penicillin and streptomycin are usually given until the reports of throat or sputum cultures can be obtained.

Chronic Conditions.

A. Chronic Bronchopulmonary Infection: Chronic bronchitis, bronchiectasis, emphysema, asthma, pulmonary fibrosis, and tuberculosis are among the commonest disorders associated with bronchial or pulmonary infection in surgical patients. Many of these patients are elderly, and smoking is often an aggravating factor. Respiratory reserve may be diminished, but is usually adequate. The major hazard is excessive bronchial secretions, with a marked tendency to postoperative atelectasis and pneumonitis.

Preoperative and postoperative management is discussed under Atelectasis (see p. 52).

B. **Diminished Pulmonary Reserve:** With the exception of thoracic procedures, pulmonary insufficiency is rarely severe enough to contraindicate necessary surgery. Diminished reserve is caused by a wide variety of disorders, particularly those mentioned above as being associated with bronchopulmonary infection. When the clinical examination shows that pulmonary function is significantly reduced, special tests should be performed (see p. 196). Preoperatively, respiratory function can often be improved somewhat by treatment of infection and bronchospasm (see above). Atelectasis and hypoxia are the chief dangers postoperatively. Acutely diminished pulmonary function after operation or injury may occasionally require tracheostomy and positive pressure breathing as a life-saving measure.

RENAL DISEASE

Chronic impairment of renal function may be fully compensated and asymptomatic under normal conditions, but renal insufficiency may develop under the adverse conditions of surgical illness. About 700 ml./day of urine are normally required for the excretion of nitrogenous wastes. In patients with damaged kidneys, considerably larger volumes of urine (1500 ml. or more) may be necessary as a result of the kidneys' inability to concentrate urine. Such patients will develop uremia if they become dehydrated or if the nitrogen load is increased, e.g., in fever and as a catabolic response to trauma or infection. Metabolic acidosis is a common additional complication (see p. 105).

The following types of renal disease are most frequently responsible for diminished reserve in surgical patients: chronic glomerulonephritis, chronic pyelonephritis, toxic nephritis, amyloid renal disease, obstructive uropathy, and acute renal failure.

Before major operations it is often advisable to evaluate kidney function by 1 or more special examinations if any of the following is present: (1) a history of renal disease, (2) age over 60, (3) diabetes mellitus, (4) hypertension, or (5) abnormal urinalysis (proteinuria, casts, low or fixed sp. gr., and oliguria or polyuria not caused by variations in fluid intake). The following special examinations are frequently of value in addition to the routine urinalysis: BUN, creatinine or NPN, PSP, a clearance test (urea or creatinine), and intravenous urograms.

Principles of Management.
  A. Preoperative Care:
    1. Evaluate renal function carefully.
    2. Correct electrolyte imbalance (see p. 99).
    3. Correct nitrogen retention by proper fluid intake and reduction of dietary protein.
    4. Correct anemia and hypovolemia by blood transfusion.
    5. Be alert for the dangerous accumulation of medications excreted by the kidneys.
  B. During Operation: The same general rules for operation and anesthesia apply as for patients with heart disease (see p. 35).
  C. Postoperative Care:
    1. Record intake and output accurately.
    2. Maintain electrolyte balance carefully with attention to

adequate fluid intake and output (see p. 99).
3. Weigh patient daily.
4. Determine BUN and creatinine postoperatively as indicated.
D. Treatment of Complications:
1. Acute renal failure (see p. 43).
2. Urinary tract infection and obstruction (see p. 375).
3. Uremia - Nitrogen retention in surgical patients is frequently reversible when precipitating factors such as dehydration and urinary obstruction are corrected. Diagnosis and treatment of these should be prompt. In the management of uremia, acidosis and other electrolyte derangements are more important than the elevation of the BUN (see p. 99).
a. Diet - Restrict protein to 0.5 Gm./Kg./day plus the amount lost in the urine.
b. Fluids - Unless the patient is anuric, give 3000-4000 ml./day and strive for a daily urine output of 1500-2000 ml. Beware of overloading and cardiac failure.
c. Electrolytes - Correct imbalances cautiously (see p. 99). The amount of sodium required to correct hyponatremia in the uremic patient may precipitate pulmonary edema. Failure to conserve fixed base causes progressive depletion which may be partially overcome by administration of the following salt mixture by mouth if oral intake is tolerated:

> ℞ Sodium citrate   100
> Calcium chloride   3
> Sig.: 2 Gm. (30 gr.) in 1 glass
>   of water t.i.d.

d. Anemia - Correct by transfusions if severe.
e. Medications -
(1) Aluminum hydroxide gel, 15 ml. q.i.d. orally, causes precipitation of soluble phosphates in the bowel and aids in reducing hyperphosphatemia. This helps to elevate serum calcium and prevent tetany.
(2) Calcium gluconate or lactate, 10%, 10 ml. I.V., p.r.n., is useful to prevent or treat tetany.

Extrarenal Azotemia.

Extrarenal azotemia is the abnormal accumulation of nitrogenous waste products in the presence of normal or potentially normal renal function. The most common cause is inadequate glomerular filtration secondary to decreased effective circulating blood volume, as occurs in shock and dehydration. BUN, NPN, and blood creatinine are elevated. After massive gastrointestinal bleeding, extrarenal azotemia may occur as a result of protein digestion and absorption plus decreased circulating blood volume. Under these circumstances the blood creatinine level is not elevated.

Since no renal disease is present, treatment is aimed at improving circulating blood volume (transfusion), electrolyte balance, and urinary output (increased fluid intake).

DIABETES MELLITUS

Well-controlled diabetes probably does not increase operative risk. However, the diabetic patient is physiologically older than his chronologic age and tends to develop atherosclerosis early; the possibility of cardiac, renal, and cerebral complications is therefore greater. The uncontrolled diabetic is also more suscep- tible to infection, which in turn aggravates his metabolic disorder For this reason the diabetic patient must be protected from even minor infections and must receive prompt antibiotic treatment when infections do occur. Furthermore, diabetes is frequently put out of control by the very conditions so often associated with surgical ill- ness: excitement and stress, anesthesia; fluid, electrolyte, and nu- tritional derangements; and inflammation. Early diagnosis and therapy are therefore essential to minimize these disturbances The collaboration of an internist in the management of the diabetes is advisable

Action of Insulin*

Product	Action		
	Onset	Maximum	Total Duration
Short-acting Insulins			
Regular, I.V.	Immediate	1 hr.	. . . .
Regular, subcut.	1/2 hr.	3 hrs.	7 hrs.
Crystalline Zinc (CZI)	1/2 hr.	3 hrs.	7 hrs.
Intermediate Insulins			
NPH	3 hrs.	8-9 hrs.	16-17 hrs.
Globin	3 hrs.		16-17 hrs.
Long-acting Insulins			
Protamine Zinc (PZI)	10 hrs.	18 hrs.	24-36 hrs.
Newer Insulins			
Semi-lente	3-4 hrs.	7 hrs.	9-10 hrs
Ultra-lente	8-9 hrs.	16-19 hrs.	24-48-96 hrs.
Lente® (30% semi and 70% ultra)	3 hrs.	8-9 hrs.	18-24 hrs.

*Adapted from Read and Baer.

Color of Benedict's Test Solution	Amount of Regular Insulin Needed
Red (2%)	10-15 units
Orange (1.5%)	5-10 units
Yellow (1%)	Up to 5 units

Elective Surgery.
A. Preoperative Care:
   1. Diet - In a well-regulated diabetic, continue the diet and insulin regimen to which the patient is accustomed. If the patient is not on an adequate diet, he should be placed on a diabetic diet and insulin dosage given q.i.d. (a.c. and h.s.) according to the urine reactions listed in the table above. A diet suitable for a normally active 70 Kg. patient should

consist of 1750 Cal.: 175 Gm. (700 Cal.) carbohydrate, 105 Gm. (420 Cal.) protein, and 70 Gm. (630 Cal.) fat.

2. Urinalysis - In every diabetic surgical patient, examine for sugar and ketone bodies before each meal and at bedtime or every 4 hours to evaluate the status of the disease.
3. Fasting blood sugar should be determined on 1 or 2 occasions before operation.
4. If infection is present, insulin therapy will often fail to eliminate glycosuria; control of infection is frequently necessary before carbohydrate tolerance will improve.
5. If diabetes is newly discovered or out of control on admission, consult an internist for assistance in regulating it.
6. Ketonuria and acidosis must be corrected prior to elective operations.

B. Day of Operation:
1. Assign the patient an early position on the operating schedule.
2. Omit modified insulin on the day of operation (and usually for several days thereafter). Perform urinalyses every 4 hours and administer regular insulin according to the color reaction (see above).
3. Continue regular oral intake up to 12 hours before surgery.
4. Patients with mild diabetes may usually be fasted preoperatively for 8-12 hours, just as is done for patients without diabetes. Patients with more severe diabetes should be given 1 L. of 5% glucose in water or saline before surgery.
5. Give fluids, electrolytes, and glucose on the day of operation according to the requirements of the operative procedure and the patient's condition.
6. When prolonged procedures are contemplated or when the patient is unable for other reasons to void at regular intervals, insert an inlying catheter. Catheterization may produce urinary tract infection and for this reason should not be done unless definitely required. Use all appropriate means to elicit spontaneous urination.

C. Postoperative Care:
1. Continue urinalyses at intervals of 4 hours and administer regular insulin according to the color reaction until the patient's accustomed diet and insulin regimen can be resumed.
2. Provide at least 150 Gm. of carbohydrate daily, either orally or by vein.
3. Do not attempt to keep the urine completely free of sugar during the first day or 2 postoperatively.

Emergency Surgery.

A. Treatment of Diabetic Acidosis Preoperatively: These patients frequently have ketonuria. If acidosis is also present (as indicated by ketonuria, red reduction for sugar in urine, and blood bicarbonate below 27 mEq./L.), treatment is urgently required along the following lines:
1. Urinalysis - Examine urine hourly for sugar and ketone bodies. (Insert an inlying catheter only if absolutely necessary.) The urine should contain some sugar at all times during intensive treatment with insulin to avoid a hypoglycemic reaction.

2. Blood chemistry - Draw blood for blood glucose, bicarbonate, sodium, potassium, and chloride. In acidosis, in addition to the losses caused by the surgical condition, there is usually acute loss of water, sodium, chloride, bicarbonate, and potassium.

3. Insulin therapy - Intensive insulin therapy is indicated. Give 50-100 units of regular insulin I.V. and a similar amount subcut. unless the patient is in shock, in which case only the intravenous route should be used. Repeat 50-75 units subcut. every 1-2 hours until there is a rapid diminution in urinary and/or blood sugar; then give insulin intravenously in glucose and saline as described below.

4. Fluids and electrolytes - Begin an infusion of physiologic saline at the outset of treatment and restore water and electrolyte balance. If blood bicarbonate is low, give sodium bicarbonate or sodium lactate by vein in calculated amounts. Marked potassium deficit is usually present and, when a urine output of 40-50 ml./hour has been established, requires correction with potassium (preferably as the phosphate), 20 mEq./L. of intravenous fluid.

5. Glucose - As soon as urinary sugar has fallen to the point where an olive or green reduction is shown on Benedict's test, change intravenous fluids to 5% glucose in saline to which is added 0.5-1 unit of insulin/Gm. of glucose (25-50 units of insulin/L.) and 20 mEq./L. potassium phosphate. The urine should contain sugar at all times to avoid hypoglycemic reactions. Continue this infusion slowly (60-70 drops/minute) throughout the surgical procedure. This will supply glucose on which the insulin can act in overcoming the acidosis.

6. Timing of operation - Unless surgical intervention (e.g., drainage of an abscess) is essential to management of the acidosis, delay operation until acidosis is controlled.

7. Postoperatively - Continue slow infusion of 5% glucose in saline or water with insulin until ketonuria has disappeared. Administer additional insulin subcut. every 1-2 hours as required to maintain slight glycosuria. Insulin dosage is based on the color of the reduction in hourly urinalyses (see p. 45). As soon as possible, shift to oral intake and give insulin every 4 hours in amounts depending upon the color reaction on urinalysis.

B. Emergency Operation for Trauma: Injured diabetic patients under insulin treatment may develop a severe hypoglycemic reaction because of failure to eat (see p. 45). Determine blood and urine sugar. If laboratory findings, history, or physical examination suggest the possibility of an insulin reaction (weakness, faintness, hunger, tremor, convulsions, or hypoglycemia), give sweetened orange juice or candy by mouth or 5% glucose in water or saline by vein. Administer 5% glucose in water or saline at 60-70 drops/minute I.V. throughout the operation. One unit of insulin per 2 Gm. of glucose may be added to this infusion. Test urine every 1-4 hours and administer regular insulin accordingly. When surgery has been completed, follow as after elective surgery.

Anesthesia. (See p. 65.)

With the aid of insulin practically any type of anesthetic may be used, but some are better than others. The following principles should be kept in mind:

A. Premedication: Keep this to a minimum. Avoid depressing the patient with narcotics unless they are definitely made necessary by severe pain. Obtain mild sedation with a barbiturate; in the older diabetic patient, use only atropine.

B. Anesthetic Agents: The anesthetist should be experienced in the problems of surgical anesthesia in diabetic patients.
   1. Local or regional procaine anesthesia is the method of choice if suitable.
   2. Spinal anesthesia is next in order of preference
   3. General anesthesia - Ether is the least desirable of these agents since it impairs the formation of glycogen and tends to increase the severity of the diabetes Cyclopropane and thiopental, with or without relaxants, are more satisfactory. Avoidance of hypoxia is essential.

OTHER FACTORS AFFECTING OPERATIVE RISK*

The Elderly Patient.

Operative risk is estimated on the basis of physiologic rather than chronologic age. The hazard of the average major operation for the patient over 60 is increased only slightly provided there is no cardiorenal or other serious systemic disease. Do not deny the elderly patient a needed operation on the basis of age alone.

A. Preoperative Studies: Assume that every patient over 60, even in the absence of symptoms and physical signs, has generalized arteriosclerosis and potential limitation of myocardial and renal reserve. In addition to routine work-up, obtain an Ecg. and BUN. Other studies should be added as indicated. Occult cancer is not infrequent in this age group, therefore, investigate suggestive gastrointestinal and other complaints even though minor.

B. Preoperative and Postoperative Precautions: ·Observe the safeguards recommended for patients with heart disease (see p. 35) and for the prevention of pulmonary (see p. 42) and thromboembolic (see p. 435) complications.

C. Medications: Aged patients generally require smaller doses of strong narcotics and are frequently depressed by routine doses. Codeine is usually well tolerated. Barbiturates often cause restlessness, mental confusion, and uncooperative behavior, and their use should therefore be limited. Preanesthetic medications should be limited to atropine or scopolamine in the elderly debilitated patient, and anesthetic agents should be administered in minimal amounts.

D. Rehabilitation: Early ambulation and return to normal eating and living habits prevent invalidism and deterioration.

*See p. 139 for evaluation of operative risk in infants and children.

The Obese Patient.

Obese surgical patients have a greater than normal tendency to serious concomitant disease and a higher incidence of postoperative thromboembolic and wound complications. Obesity also increases the technical difficulties of surgery and anesthesia. For these reasons, it may at times be advisable to delay elective surgery while the patient loses weight on a reducing diet

Pregnancy.

Elective major surgery should be avoided during pregnancy. However, normal, uncomplicated pregnancy has no debilitating effect and does not alter operative risk except as it may interfere with the diagnosis of abdominal disorders and increase the technical problems of intra-abdominal surgery Abortion is not a serious hazard after operation unless peritoneal sepsis or other significant complication occurs. During the first trimester, congenital anomalies may be induced in the developing fetus by hypoxia. It is preferable to avoid surgical intervention during this period; if surgery does become necessary, the greatest precautions must be taken to prevent hypoxia and hypotension. The second trimester is usually the optimum time for operative procedures.

PREOPERATIVE AND POSTOPERATIVE ORDERS

Preoperative Notes.

On the day preceding operation, all details of the patient's work-up should be reviewed and a "preoperative note" written in the chart. The purpose of this note is to give the basis for the diagnosis and the reasons for the proposed operation. It also serves as a final check on the adequacy of the preoperative management.

Preoperative Orders.

A. Skin Preparation: Designate the specific region to be prepared.
B. Blood Transfusions: If transfusions may be required during operation, arrange for a sufficient number of units to be cross-matched and available.
C. Enema· Enemas should not be given routinely. The following are indications for an enema on the evening before surgery:
   1. Operations, chiefly abdominal, likely to be followed by significant paralytic ileus and delayed bowel function.
   2. Operations on the colon, rectum, and anal region.
   3. Constipation.
D. Diet: Omit solid foods for 12 hours and fluids for 8 hours preoperatively. Special orders are written for diabetics (see p. 45) and for infants and children (see p. 139).
E. Bedtime and Preanesthetic Medication: See p. 66.
F. Nasogastric Tube: Operations on the gastrointestinal tract are the chief indication for intubation, usually in the morning just before operation, to control postoperative ileus.
G. Inlying Catheter: A Foley catheter is inserted for constant bladder drainage if urinary retention is expected postoperatively or if bladder distention during operation will interfere with exposure in the pelvis (e.g., during a combined abdominoperineal resection).

H. Venous Cutdown: Operations associated with marked blood loss require the placement of an intravenous cannula or polyethylene tube preoperatively for rapid blood replacement.

I. Miscellaneous: Complex operations may require additional preparation.

Postoperative Orders.

Postsurgical patients should be accompanied by a physician or other adequately qualified attendant while en route from the operating room. Detailed orders should be sent with each postoperative patient, and the nurse receiving him should be given a verbal report on his condition. The postoperative orders should provide for appropriate observations and treatment, which fall into the following categories:

A. Special Observations:

   1. BP, pulse, and respiration should be recorded at regular intervals after all major operations (e.g., every 15-30 minutes until stable, then hourly). A significant drop (specify) in BP should be reported to the surgeon immediately.

   2. Under certain conditions, orders should be given to observe closely for such developments as respiratory distress, cyanosis, wound bleeding or drainage, and inadequate circulation distal to a cast or traction tie.

B. Position in Bed: Designate specifically, e.g., flat or on side, sitting (semi-Fowler's), or head low (Trendelenburg).

C. Mobilization: Prescribe bed rest, standing to void, or ambulation. After general anesthesia, the patient should be turned from side to side every 30-60 minutes until he is able to turn himself. Hyperventilation, coughing, and active motion of the feet and legs every 1-3 hours during the working hours should be required until the patient is ambulatory.

D. Intake and Output: Record the fluid intake and output for at least 24 hours after major surgery and as long thereafter as necessary for control of fluid balance. Order either nothing by mouth or a specific diet. List the parenteral fluids, if any, to be administered during the first 24 hours. Patients with fluid balance problems should be weighed daily. Place an order for management in case the patient is unable to void within a specified period (usually 6-8 hours) after operation.

E. Drainage Tubes: If a nasogastric tube is used, it should be connected to suction and irrigated every 1-2 hours with 15-30 ml. of saline. Urethral, chest, biliary, and other drainage catheters call for specific orders.

F. Medications: These may include narcotics, sedatives, antibiotics, antiemetic drugs, etc. Always resume essential preoperative medications such as digitalis and insulin.

G. Oxygen Therapy: See p. 84.

H. Special Laboratory Examinations: During the first 24 hours it may be necessary to obtain such studies as Hct., Hgb., or CBC, urinalysis, blood chemistries, blood volume determinations, and x-rays.

POSTOPERATIVE COMPLICATIONS

OPERATIVE WOUND COMPLICATIONS

Wound Disruption.

An incidence of wound rupture of up to 3% in laparotomies has been reported, but the frequency can be kept well under 1%. Vertical abdominal wounds are slightly more vulnerable to dehiscence than transverse wounds. Disruption may occur as late as a month postoperatively, but is commonest between the fourth and twelfth days.

   A. Predisposing Factors: The major cause is poor surgical technic in wound closure. Malnutrition, vitamin C deficiency, hypoproteinemia, anemia, obesity, drains, postoperative cough, vomiting, distention, premature removal of sutures, and all disorders which put an undue strain on the wound may predispose to wound disruption.

   B. Sudden serosanguinous drainage from the wound is practically pathognomonic of wound rupture. Occasionally the patient is conscious of a tearing sensation as the suture line gives way during coughing or straining. Rarely, a loop of bowel or the omentum will prolapse through the wound. There is little pain or reaction unless extensive evisceration takes place.

   C. Prophylaxis: Predisposing conditions should be corrected or prevented as far as possible. Drains and colostomies should be brought out through separate stab wounds when feasible. The major incision in clean operations should be closed in layers with interrupted nonabsorbable sutures, approximating fascial structures under proper tension. In addition, retention sutures should be placed through all layers of the abdominal wall if wound closure is precarious.

   D. Treatment:
   1. Minimal disruption - Using aseptic technic, hold the wound edges together with strips of flamed adhesive tape, apply a sterile dressing and an abdominal binder, maintain bed rest, relieve abdominal distention with nasogastric suction as required, and treat the systemic condition. This procedure is applicable only when the sole evidence for dehiscence is slight serosanguinous drainage without significant wound separation.

   2. Major disruption - Resuture the entire wound with through-and-through wire, nylon, or silk sutures, under general anesthesia if necessary. Primary healing usually takes place.

   E. Prognosis: Mortality after wound dehiscence is about 10% and is due primarily to the patient's underlying disease. The incidence of hernia following through-and-through closure of disrupted wounds is also about 10%.

Wound Infection.

Postoperative wound infection is usually apparent within the first week after operation, but its appearance may be delayed, especially if antibiotics are given. Infection most commonly occurs

in the fat and subcutaneous tissues and is not a significant factor in wound disruption. The development of fever 4-7 days postoperatively is frequently due to wound infection. Treatment consists of early drainage and appropriate anti-infective therapy.

PULMONARY COMPLICATIONS

Atelectasis.

Obstruction of a bronchus or bronchiole with resultant collapse of a portion of the lung occurs after 10-20% of major operations and is the most frequent complication of thoracic surgery. Involvement of multiple, small patchy areas is most common, but massive atelectasis of an entire lobe or lung may occur. Onset usually occurs within the first few days postoperatively, and is almost invariably associated with pneumonitis in the collapsed area.

A. Predisposing Factors:
1. Preoperative - Atelectasis is 4 times as frequent in patients over 60, and twice as frequent in males. The rate of occurrence is higher when the patient is 30% or more above ideal weight. Chronic bronchitis, bronchiectasis, asthma, and smoking predispose to increased secretions and a higher incidence of atelectasis and pneumonitis postoperatively. For similar reasons, acute respiratory infection definitely increases the incidence and severity of pulmonary complications.
2. Operative - Hypoventilation, accumulation of bronchial secretions, aspiration of gastric contents, and prolonged immobilization during operation set the stage for the development of atelectasis postoperatively.
3. Postoperative - Inadequate ventilation, failure to change position, and failure to remove tracheobronchial secretions by cough or aspiration are the most important factors. Depressant doses of narcotics, abdominal distention, wound pain, and general debility are aggravating conditions.

B. Diagnosis: Higher than anticipated fever in the first few days after operation is frequently the earliest sign of atelectasis. Temperature may rise suddenly, reaching 102° F. (38.9° C.) or more. Cough and sputum may not be present at the onset, but usually develop within 1-2 days. Diminished breath sounds and bronchial breathing with scattered moist rales are heard over the area of collapse. When a major portion of the lung is atelectatic, the mediastinal structures shift to that side and deviation of the trachea may be apparent at the jugular notch. Dyspnea, cyanosis, and prostration indicate extensive atelectasis and pneumonitis.

C. Treatment: Examine the chest of postoperative patients once or twice daily during the first week after surgery. Act promptly on the earliest signs of atelectasis since this disorder is often quickly reversible if treated immediately. Obtain a chest x-ray and sputum culture at the beginning of treatment in severe cases.

Vigorous coughing, deep breathing, and change of position

at intervals of 30 minutes to several hours are the most im-
portant preventive and therapeutic measures. Early ambula-
tion is also of value. If these measures are inadequate (as
they frequently are in older patients), tracheal aspiration
should be carried out as often as necessary.

1. Voluntary coughing - Insist on a short period of hyperven-
   tilation and forceful coughing every hour. Close supervision
   and encouragement are essential. Cough and cooperation
   will be improved by administration of a narcotic to relieve
   wound pain and apprehension   The expectoration of thick,
   yellowish, tenacious plugs of sputum is usually necessary
   to bring improvement.

2. Tracheal aspiration - If voluntary coughing is inadequate,
   aspirate the trachea as often as required to clear secretions.
   Tracheal catheterization is best accomplished with the pa-
   tient in a sitting position and the tongue held forward by an
   assistant. A 16-20 F. catheter (connected to suction with
   a "Y" tube so that suction may be interrupted) is then
   passed through the nostril into the pharynx. The patient is
   then instructed to take a few deep breaths, and the catheter
   is advanced quickly into the trachea during inspiration.
   Paroxysmal cough and loss of voice indicate that the catheter
   is in correct position. The catheter is then moved up and
   down the trachea for aspiration. Suction is interrupted as
   necessary to allow the patient to rest and breathe.
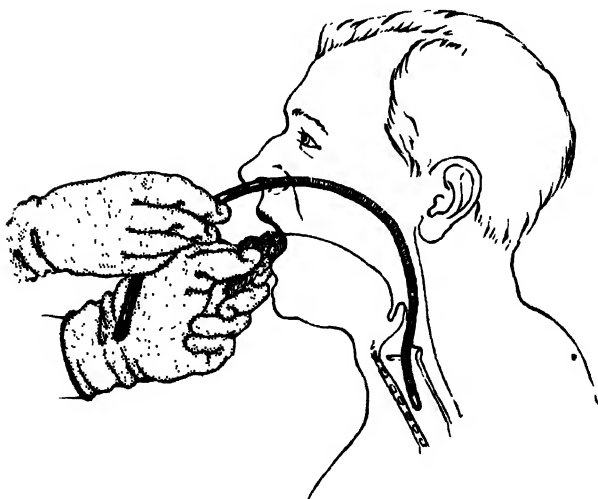
Technic of Tracheal Aspiration

If this method fails, bronchoscopy may be necessary. Usually, however, tracheostomy (see p. 166) is advisable in patients in whom tracheobronchial secretions cannot be adequately removed by transnasal tracheal aspiration.

3. Bronchoscopic aspiration - This may occasionally be necessary at the conclusion of an operation to remove foreign material or plugs of mucus from the bronchi. Thereafter, tracheostomy is more effective than bronchoscopy and will usually be preferable.

4. Tracheostomy - In debilitated patients unable to raise secretions and in cases in which tracheal aspiration fails or there is significant respiratory embarrassment, tracheostomy should be done without delay (see p. 166).

5. Intermittent positive pressure breathing (IPPB) - This type of breathing for 3-5 minutes every hour is very useful. The Bennett valve or similar apparatus is employed. The oxygen should be humidified by nebulization of physiologic saline solution or a wetting agent (Alevaire®).

6. Inhalations -

  a. Steam - Inhalation of warm, moist air is soothing to a dry, inflamed tracheobronchial tree. The steam kettle is the traditional source. One tsp. of Compound Tincture of Benzoin, U.S.P., may be added to each quart of water. This produces a pleasant medicinal odor but does not increase the efficacy of the steam.

  b. Aerosol therapy -

    (1) Nebulizers - These produce fluid particles smaller than 8-10 microns in diameter. The Vaponephrine® model and the DeVilbiss No. 40 are satisfactory for intermittent use. For continuous aerosol therapy, apparatuses such as Mist-O_2-Gen® and Humidox® are available and may be placed inside an oxygen tent.

    (2) Water or saline - Nebulization of water or 1-3% saline solution is more satisfactory than steam inhalations and should be used with oxygen by nasal catheter or face mask to reduce irritation.

    (3) Surface tension lowering agents - Alevaire® contains glycerin, sodium bicarbonate, and the detergent superinone and, like ethyl alcohol, is thought by some to be helpful in liquefying bronchial secretions when nebulized. The value of these medications has not been proved.

    (4) Enzymes - Trypsin (Tryptar®) and desoxyribonuclease (Dornase®) have been suggested for use in lysing thick bronchial secretions, but reactions occur and enzyme aerosol therapy is not advised.

    (5) Bronchodilator drugs - These are indicated when bronchospasm contributes to cough and respiratory distress, as in asthmatic bronchitis, which may be exacerbated by surgical illness. An aminophylline rectal suppository every 6-12 hours may be of value when prolonged bronchodilator effect is desired.

      One of the following may be administered by nebulization:

(a) Isoproterenol Hydrochloride Inhalation, U.S.P. (Isuprel®, Aludrine®), usually 3-7 inhalations of 1:100 solution or 5-15 inhalations of 1:200 solution with hand nebulizer; with oxygen aerosolization, give no more than 0.3 ml. of 1:100 solution or 0.5 ml. of 1:200 solution in a 15-20 minute period. Repeat every 4 hours p.r.n. or more often in severe cases. Simultaneous use of epinephrine is contraindicated.

(b) Epinephrine Inhalation, U.S.P., 0.5-1 ml. of a 1:100 aqueous solution by inhalation every 2-4 hours. Use cautiously in hypertensive and elderly patients.

(6) Antibiotics - Inhalation of antibiotics is not advisable because the patient may become sensitized to the drug. Use systemic antibiotics if anti-infective therapy is necessary.

c. Carbon dioxide and oxygen - Inhalation of a mixture of 5% carbon dioxide and 95% oxygen at a rate of 5-7 L./ minute by means of a mask or nasal catheter for 10-15 minutes 3-4 times daily tends to improve depth of respiration and has some bronchodilator effect. These actions make cough more effective, clear out secretions, and thus relieve chronic coughing. This regimen may be particularly useful in prevention or treatment of postoperative atelectasis.

7. Antibiotics - Pneumonitis should be treated with penicillin or a tetracycline (see p. 614) until sputum culture and sensitivity studies are available.

8. Expectorants - See p. 610.

9. General measures - Treat all contributory conditions, such as abdominal distention (see p. 57). Change the patient's position hourly and encourage early ambulation. Respiratory exchange and coughing are most efficient in the semi-sitting position.

D. Prophylaxis:

1. Preoperative measures -

   a. Never perform elective surgery in the presence of an acute respiratory tract infection.

   b. Treat chronic respiratory disorders such as bronchitis, bronchiectasis, and asthma in order to reduce infection and sputum volume and to improve ventilatory capacity.

      (1) The patient should stop smoking.

      (2) Give aerosol therapy with a bronchodilator drug and a wetting agent 4 times daily by IPPB.

      (3) Penicillin administration in bronchiectasis combined with postural drainage will frequently reduce sputum volume markedly.

      (4) Repeated pulmonary function tests may be helpful in evaluating effectiveness of treatment.

2. During operation - Expert anesthesia management is the most important feature.

3. Postoperative measures -

   a. Postoperative orders should include instructions for turning, hyperventilating, and coughing.

   b. If chronic respiratory tract disease was present preoperatively, institute IPPB and aerosol therapy postoperatively as for established atelectasis.

Pneumonitis and Bronchial Pneumonia.

These pulmonary infections are usually secondary to atelectasis and respond to treatment for that condition. When they occur independently, the principal treatment is with an antibiotic drug chosen on the basis of sputum culture and sensitivity studies. After specimens have been obtained, penicillin may be used empirically until reports of these tests are available (see p. 614).

Pulmonary Embolism and Infarction.

Pulmonary emboli occur following 0.1-0.2% of operations. They are more common after middle age, in women, and following operations on the abdomen, pelvis, and lower extremities. Other predisposing factors include obesity, cardiac disease, and general debility. Pulmonary embolism is a complication of thrombophlebitis, usually of the leg veins, but embolism frequently precedes the clinical appearance of vein involvement by 1-3 days. In about 40% of cases, no evidence of thrombophlebitis will ever be found. Pulmonary emboli are usually not fatal, and the transient effects may go unrecognized or may be misdiagnosed as atelectasis or pneumonia. It is important to realize that small emboli precede fatal ones as a warning in one-third of cases. Prompt recognition and treatment of all emboli is therefore essential.

A. Diagnosis: The cardinal symptom of the small infarct is a sudden onset of pleuritic pain, occasionally associated with blood-streaking of sputum. The following additional symptoms and signs may or may not develop: friction rub, cough, and localized signs of pulmonary consolidation. Larger emboli cause dyspnea, cyanosis, and prostration; if a major pulmonary artery is obstructed, sudden death may occur as a result of acute cor pulmonale. A concomitant rise of temperature, pulse, and respiration is often seen on the clinical chart immediately following an infarct. X-ray of the chest will usually show an area of consolidation after 24-48 hours. This area is commonly (but not always) in the periphery of the lung and roughly in the shape of a triangle, with its base at the lung surface. A small amount of pleural fluid may be seen.

B. Treatment:
   1. Anticoagulants - Begin anticoagulant therapy with heparin **as soon as a pulmonary embolus is suspected.** The typical sudden occurrence of pleuritic pain which cannot be otherwise accounted for is sufficient indication for anticoagulant therapy even in the absence of other signs and symptoms; thrombophlebitis of the leg veins may be assumed and therapy planned accordingly (see p. 433).
   2. General measures - Symptomatic treatment includes narcotics for pain and oxygen therapy for dyspnea.
   3. Surgical measures -
      a. Vein ligation - Ligation of both superficial femoral veins or of the distal vena cava should be considered when pulmonary emboli (presumed to arise in the legs) occur during anticoagulant therapy or when anticoagulants are

contraindicated.

   b. **Embolectomy** - Rarely, a massive and potentially lethal pulmonary embolus may be recognized under circumstances permitting emergency thoracotomy and removal of the embolus from the pulmonary artery.

C. **Prophylaxis:** The prophylaxis of pulmonary embolism depends upon the prevention of thrombophlebitis (see p. 433).

ABDOMINAL COMPLICATIONS

Paralytic Ileus.

   See p. 274.

Acute Gastric Dilatation.

   Acute gastric dilatation is caused by a reflex inhibition of the stomach. It occurs most commonly early in the postoperative period after major surgery or severe trauma.

A. Diagnosis· Repeated regurgitation of small quantities of thin, dark, brownish-green or black fluid with a foul (but not fecal) odor is characteristic. Copious vomiting is rarely seen. Several liters of fluid may accumulate in the stomach so that the patient becomes hypovolemic and hypochloremic. The appearance may be that of shock, and failure to relieve distention promptly at this stage may be fatal. Diagnosis is confirmed by observing the distended, tympanitic stomach in the epigastrium.

B. Treatment: Pass a nasogastric tube and aspirate the stomach immediately if gastric dilatation is suspected. Relief is usually dramatic. Correct electrolyte and fluid imbalance. Continue nasogastric suction and parenteral alimentation until gastric emptying resumes.

Subphrenic Abscess.

   Subphrenic abscess is secondary to intra-abdominal sepsis and occurs most frequently after a perforated appendix or peptic ulcer. The right subphrenic space is the commonest site.

A. Diagnosis: There is almost invariably a history of intra-abdominal operation or sepsis. Fever and leukocytosis of unexplained origin should arouse suspicion of a subphrenic abscess in such a patient. Localizing symptoms and signs are usually slow to appear and may be further delayed by antibiotic therapy.

   1. Symptoms and signs - Pain, tenderness, and occasionally edema of the skin develop in the lower thoracic region overlying the abscess. Pain may be referred to the top of the shoulder due to diaphragmatic irritation. There may be signs of fluid or basilar atelectasis and limitation of diaphragmatic motion on the side of the lesion. Pleural effusion may be present. The liver may be displaced downward and may appear to be enlarged. Septic fever and leukocytosis will be present. Rupture of the abscess into the pleural cavity usually results in massive pyopneumothorax with dyspnea and marked toxemia. Rupture into the bronchial tree results in a lung abscess with copious expectoration of pus.

2. X-ray findings - Diaphragmatic motion on the side of the lesion is usually limited or absent. Elevation or upward bulging of the diaphragm and a small pleural effusion are often noted. A fluid level in an abscess cavity beneath the diaphragm is pathognomonic but is not always present.

B. Treatment: Surgical drainage by an extraperitoneal and extrapleural approach is the only treatment of value. Broad-spectrum antibiotic therapy should be employed, but only as an adjunct. Exploration of the subphrenic space on the basis of suspicion of abscess may be necessary and is justifiable to avoid the serious complications which follow diaphragmatic perforation.

C. Prophylaxis: Early recognition and prompt surgical treatment of predisposing lesions, such as appendicitis and perforated peptic ulcer, and intensive antibiotic therapy of all forms of intra-abdominal sepsis have, in recent years, greatly reduced the incidence of subphrenic abscess.

URINARY TRACT COMPLICATIONS*

Acute Postoperative Retention.

A. Etiology: Inability to void after operation is most often due to (1) reflex spasm of the sphincters, caused by pain or anxiety; (2) paralysis of the detrusor mechanism, caused by operations within the pelvis; and (3) vesical neck obstruction by an enlarged prostate. The incidence of acute postoperative retention is highest following rectal, perineal, and abdominal surgery.

B. Diagnosis: Failure to pass urine postoperatively may be simply the result of inadequate fluid intake with oliguria. More rarely the cause will be acute renal failure. If the patient is unable to void in spite of an urgent desire to do so, or if his bladder is palpable above the pubis, he probably has acute retention. A deeply sedated patient may be unaware of a distended bladder postoperatively. NOTE: Because an over-distended bladder becomes atonic, in which case prolonged catheter drainage will probably be required, no patient should be allowed to go more than 8 hours postoperatively without an evaluation of his inability to void.

C. Treatment: Catheterization is followed by cystitis in about 20% of cases. Therefore, try simpler measures first.

1. Relieve acute pain and anxiety by a narcotic or sedative and reassurance.

2. Have the patient sit on a commode or stand to void. Warm the bed pan if one must be used.

3. Turn on a water faucet to encourage urination.

4. Give Bethanechol Chloride Injection, U.S.P. (Urecholine®), 2.5 mg. subcut. ($^1/24$ gr.), followed in one-half hour by 5 mg. ($^1/12$ gr.) subcut. if there have been no untoward side effects and spontaneous urination has not occurred. This drug should never be given in the presence of organic obstruction of the urinary or gastrointestinal tract; whenever increased peristalsis might prove harmful; or in asthma or

*Urinary infection, see p. 375; acute renal failure, see p. 43; uremia, see p. 43.

thyrotoxicosis. Mild side effects such as abdominal cramps, flushing, and sweating occur occasionally even with small doses.

5. Catheterization - Catheterize when other measures fail and before marked bladder distention (over 500 ml.) occurs. If a third catheterization is required, insert a Foley catheter and leave the patient on constant bladder drainage until he is ambulatory or for at least 4-5 days. Patients who are unable to void postoperatively because of an enlarged prostate may require prostatectomy before they can regain their power to urinate spontaneously.

If urinary tract infection follows catheterization, obtain a urine culture and perform antibiotic sensitivity studies. Begin antibiotic therapy as indicated after the catheter has been removed (see p. 614).

D. Prophylaxis: Examine preoperatively for prostatic hypertrophy, cystocele, and other disorders likely to cause postoperative retention. When such defects are found they should be corrected before operation if possible; otherwise the patient should be watched carefully so that overdistention of the bladder postoperatively can be avoided. A Foley catheter should be inserted preoperatively on all patients scheduled for extensive pelvic operations, and bladder drainage should be continued after surgery until the patient is fully ambulatory.

ACUTE PAROTITIS

Acute parotitis as a complication of surgery is now rare because of the declining incidence of such predisposing disorders as dental sepsis and postoperative dehydration. It is most likely to occur in a debilitated, febrile, dehydrated, elderly patient with poor oral hygiene, or in patients on diuretic therapy or anticholinergic drugs.

Swelling and tenderness appear in the parotid gland at the angle of the jaw, accompanied by trismus, systemic toxicity, tachycardia, leukocytosis, and fever sometimes rising to 104°F. (40°C.). Bilateral involvement can occur. Pus may sometimes be expressed from a swollen, inflamed Stensen's duct. Abscesses may form within the gland, but fluctuation is late because of the deep location and trapping by fibrous septa and tense capsule.

X-ray treatment is effective in aborting the process if carried out immediately after onset. Established parotitis will usually respond to penicillin, local hot packs, mouth care, and restoration of fluid balance. When abscesses develop, drainage is required; incisions must be made parallel to the facial nerve to avoid injuring it.

Preoperative correction of dental sepsis and good postoperative oral hygiene and adequate fluid intake are effective preventive measures.

COMPLICATIONS OF BLOOD COAGULATION

Diagnosis of Bleeding and Coagulation Disorders.

Bleeding and coagulation disorders are usually discovered because of (1) a history of abnormal bleeding or a predisposing condition, such as liver disease; or (2) the unexpected occurrence of excessive hemorrhage at operation or after trauma. It is essential in surgical practice to recognize the presence or the possibility of a clotting defect before surgical intervention so that diagnostic and corrective measures can be carried out. Inadequate hemostasis is the commonest cause of postoperative bleeding. Rule out this possibility before assuming that a hemorrhagic disorder is present.

A. Screening Measures:
1. History - The most important clues to the existence of a clotting disorder are found in the history. Every patient should be questioned preoperatively regarding the following conditions, any of which may be associated with a coagulation dyscrasia·
   a. Personal or family history of abnormal bleeding, bruising, or purpura
   b. Unusual hemorrhage in connection with operation or dental extraction.
   c. Liver disease.
   d. Ingestion of drugs capable of depressing prothrombin, e.g., salicylates and intestinal antiseptics (neomycin, succinylsulfathiazole, etc).
2. Routine bleeding and clotting times - These are of little value in detecting occult clotting defects. Chief reliance should be placed on the history and thorough laboratory investigation of all questionable cases.

B. Laboratory Studies· Patients suspected of having a bleeding or coagulation defect should be investigated with appropriate laboratory tests (e.g., bleeding time, tourniquet test, platelet count, coagulation time, clot retraction, prothrombin time, and prothrombin consumption). Accurate diagnosis of the specific factors involved in coagulation disorders will often necessitate additional special procedures. Consultation with a hematologist or clinical pathologist is essential in complicated cases.

Emergency Treatment of Abnormal Bleeding.

Abnormal bleeding at operation or in the postoperative period is a rare but potentially dangerous complication. Among the most frequent of the causes which must be considered are liver disease (hypoprothrombinemia), hemolytic transfusion reaction, massive transfusion of citrated blood or dextran, fibrinogen-fibrinolysin disorders, and hemophilia. Always rule out inadequate hemostasis.

A. Diagnosis Establish the diagnosis at once, if possible, so that specific treatment may be given. Obtain blood for coagulation studies. An immediate report should be available on platelet count, coagulation time, and prothrombin time. When transfusions have been given, verify the accuracy of the cross-match if in doubt. Consultation with a hematologist is advisable in all obscure cases.

B. Treatment: Specific diagnosis often cannot be made promptly. If bleeding is serious, it is then necessary to try 1 or more

of the following measures empirically:

1. Fresh blood or plasma - Administer 500 ml. or more of blood or plasma drawn within 6 hours in siliconized or plastic containers. Blood processed in this manner contains platelets. There are no platelets in plasma unless it has been specially prepared as "platelet-rich" plasma. Fresh plasma (or blood) provides prothrombin, fibrinogen, antihemophilic globulin, factor V, and other factors which may be deficient in the patient's own blood.

2. Vitamin K - Give Phytonadione, U.S.P. (vitamin K_1, Mephyton®), 50-100 mg ($3/4$-$1^1/2$ gr.) I.V. slowly (no faster than 10 mg./minute). This agent is of specific value in hypoprothrombinemia.

3. Corticosteroids - If the platelet count is low or if a drug reaction is suspected, give Hydrocortisone Sodium Succinate, N.N.D. (Solu-Cortef®), 100 mg. I.V

4. Calcium - Depletion of calcium may result from massive transfusion of citrated blood. Prevent or treat this by injecting 1 Gm. (15 gr.) of calcium chloride or calcium gluconate (10 ml of 10% solution) I.V. slowly after every 2000 ml. of blood.

5. Fibrinogen - Prompt, intensive therapy may be necessary in fibrinogen-fibrinolysin disorders. These are very uncommon conditions which may occur in carcinoma of the prostate, a variety of acute stress conditions, and in complications of pregnancy. When fibrinolysis is the cause of bleeding, the patient's clot will usually lyse completely within a few hours at 98.6°F. (37°C.). Plasma or whole blood is indicated. If ineffective, give concentrated fibrinogen (Parenogen®), 3 Gm. (45 gr.) I.V. in 150 ml of diluent and repeat p.r.n.

6. Topical thrombin - The topical application of thrombin may be an aid in controlling surface oozing.

NONSPECIFIC SYMPTOMS

FEVER

Reduction of Fever by Nonspecific Means.

Of primary importance in the control of fever is the removal of the primary cause whenever possible. In general, body temperature above 105°F. (40.6°C.) rectally persisting more than a few hours should be reduced by nonspecific methods if fever is the chief cause of distress or is associated with cerebral symptoms and if it can be reduced without complications.

A. Surface Applications:

1. Ice or cold water - Cold sponges or baths and the application of ice caps produce surface cooling and make the patient more comfortable.

2. Alcohol, 70% - Sponging of the trunk and extremities results in cooling by evaporation of the alcohol.

3. Baths - Warm or tepid baths cause peripheral vasodilatation with external loss of heat.
4. More radical methods of reducing temperature, including ice-water enemas and refrigerated blankets, are rarely necessary.

B. Antipyretic Drugs: (See p. 605.) These drugs have both an antipyretic and an analgesic effect. They act on the heat-regulating mechanism of the CNS to cause a greater dissipation of body heat through cutaneous vasodilatation. They may obscure the clinical picture or cause undesirable side-effects, especially diaphoresis, nausea and vomiting, or, less commonly, skin eruptions, hematologic changes, and cardiovascular depression.

NAUSEA AND VOMITING

Relief of nausea and vomiting is essential to the patient's comfort and to the prevention of fluid imbalance. The underlying cause should be treated promptly if possible. Symptomatic measures include bed rest, withholding of oral intake, and administration of an antiemetic or sedative drug.

HICCUP (SINGULTUS)

Although hiccup is usually self-limited (disappearing within a few minutes to an hour), it can be sufficiently persistent and exhausting to endanger life in a debilitated patient. It may be produced by any condition which irritates the afferent or efferent phrenic nerve pathways. The etiology is therefore quite varied: CNS, cardiopulmonary, or gastrointestinal disorders, renal failure, infectious diseases, etc. Treatment should be directed at the cause when possible, but therapy must frequently be symptomatic.

Treatment.
A. General Measures: Breath-holding, drinking a large glass of water, or gastric lavage with a warm 1% solution of sodium bicarbonate may be effective. Rebreathing into a paper bag or administration of 10-15% CO_2 by face mask induces hyperventilation and may interrupt the reflex. Inhalation of Amyl Nitrite, U.S.P., from a broken ampul will occasionally terminate the attack after a few breaths. Tranquilizing drugs such as chlorpromazine hydrochloride (Thorazine®) or triflupromazine hydrochloride (Vesprin®) are worthy of trial in prolonged hiccup (see p. 622). Barbiturate sedation (see p. 607) may also be tried.
B. Surgical Measures: If conservative therapy fails, phrenic nerve interruption by procaine block or by crushing should be considered.

FLATULENCE

Flatulence may be due to dietary causes and functional and organic disease of the digestive system. Eliminate specific causes. Gastrointestinal gas consists largely of swallowed air.

Treatment.
  A. Correction of Aerophagia: Explain the etiology to the patient and gain his cooperation. Emotional tension may be an underlying cause and should be treated.
  B. Diet:
     1. Avoid overeating, eating too fast, and eating while under emotional strain. Chewing gum, laxatives, and dietary indiscretions aggravate the condition.
     2. Prescribe a bland diet (see p. 95) and restrict gas-producing and irritating foods.
  C. Relieve constipation: See below.
  D. Sedative or anticholinergic drugs may have value but usually act only as placebos.

CONSTIPATION

Constipation is a common postoperative complaint. Resumption of normal bowel habits is encouraged by early ambulation, bathroom privileges, discontinuance of narcotics, and normal oral intake.

Treatment.
  A. Laxatives (see p. 610)· A mild laxative (e.g., liquid petrolatum, milk of magnesia, methylcellulose, or dioctyl sodium sulfosuccinate) is frequently helpful in the postoperative period, but should usually be withheld until peristalsis has returned and oral intake begun. Contraindications to laxatives include peritoneal or bowel inflammation, paralytic ileus, and mechanical intestinal obstruction.
  B. Enemas: Enemas should be used only as temporary measures to combat induced constipation and fecal impaction; to cleanse the bowel for diagnostic studies and surgery; and to aid in the restoration of normal bowel habits.
     1. Warm tap water (nonirritating), 500-1500 ml. (1-3 pints). Contraindicated in congenital megacolon because of the danger of excessive water absorption. Use a saline enema instead.
     2. Saline enema (nonirritating), 500-1500 ml. (1-3 pints) of warm physiologic saline.
     3. Soapsuds (S.S.) enema (irritating), 500-1500 ml. White toilet soap is stirred into warm tap water until it is opalescent.
     4. Hypertonic phosphate enema (irritating) consists of saturated solution of sodium phosphate. Commercially available disposable enema kits containing 4-5 oz. of this solution are very convenient (Travad® Enema, Clyserol®, and Fleet® Enema).
     5. Oil Retention Enema: 180 ml. of mineral oil or, preferably, cottonseed oil is introduced to soften impacted feces or in

the management of acutely painful rectal lesions Over-
night retention of the oil followed by an S.S or hypertonic
phosphate enema in the morning is often advantageous.

C. Fecal Impaction: When severe constipation or scanty, inter-
mittent diarrhea develops in a bedridden or sedentary patient,
fecal impaction should be suspected and a rectal examination
performed. Elderly and debilitated patients and those taking
narcotics are most susceptible. Fecal impaction is prevented
by the judicious use of anticonstipation measures, including
laxatives and enemas. Treatment consists of an oil retention
enema followed by a cleansing enema Manual removal of the
impaction is not infrequently necessary.

DECUBITUS ULCER
(Pressure Sore; Bedsore)

Decubitus ulcers are caused by sustained pressure on the skin,
usually over bony prominences such as the sacrum, ischium, tro-
chanter, and heel. They occur quite commonly in bedridden pa-
tients who are weak, aged, malnourished, or paralyzed and who
are receiving poor nursing care. Unrelieved pressure of only a
few hours may be sufficient to produce a decubitus ulcer in a sus-
ceptible individual. Decubiti characteristically begin as a small
area of redness and tenderness which soon breaks down to form an
indolent ulcer unless protected from further pressure. In neglected
cases, large defects in skin and soft tissues may result from the
combined effects of pressure, infection, and poor healing power
Osteomyelitis of the underlying bone may occur.

Treatment.
A. General Measures: Relieve pressure by frequent change of
position and protection of the involved area, if necessary, by
pillows, pads, and rubber rings. Bed clothing and skin must
be kept clean and dry. Correction of malnutrition and anemia
and control of infection are often essential to healing.
B. Local Measures: Decubitus ulcers should be kept clean, well-
drained, debrided, and either exposed or covered with dry
sterile dressings. Enzymatic debridement may occasionally
be advisable (see p. 136). Topical applications have little
value. Invasive local infection is treated by drainage, saline
compresses, and systemic antibiotics as indicated.
C. Surgical Treatment· Surgical treatment consists of complete
debridement, including removal of any bony prominences or
sequestra, and closure of the wound by a local rotation flap
This will provide an adequate pad over the bone and avoids su-
ture lines over the critical area of pressure. The donor area
may frequently be closed by direct approximation, but in some
cases a split-skin graft may be required.

Prophylaxis.
The most important elements in prevention are good nursing
care, early mobilization, and maintenance of good nutrition. Bed-
ridden patients should be inspected frequently for areas of skin
damage which might progress to ulceration. An alternating pres-
sure pad or foam rubber mattress is useful.

3 . . .

Anesthesia

Anesthetic agents and technics are usually employed to facilitate surgical procedures. In the form of "nerve blocks" they may also be used for diagnostic and therapeutic purposes.

The conduct of a safe anesthetic procedure begins with the preoperative visit to the patient's bedside and ends after the patient has completely recovered from the anesthetic and any postanesthetic complications.

Anesthetic management thus includes· (1) Evaluation and preparation of the patient; (2) familiarity with the action of the drugs to be used; (3) ability to use the recommended technics; (4) ability to administer supportive measures as necessary, e.g., maintenance of adequate pulmonary ventilation; and (4) care of the patient in the postanesthetic period.

EVALUATION OF THE PATIENT

Except in emergencies, patients should be examined by the anesthetist on the day before the proposed procedure. A thorough review of the history, physical examination, and laboratory findings is essential to ensure proper preparation of the patient, to aid in the selection of the agent and type of anesthetic procedure, to anticipate complications, and to suggest postanesthetic care

History.
In taking the preanesthetic history special emphasis should be placed on the cardiorespiratory system. Question the patient about the following:
- A. History of Previous Anesthesia: Agents and methods which have caused difficulties or complications can be avoided or safeguards taken.
- B. History of Drugs Used by the Patient· The following groups are of importance.
  - 1. CNS depressants - Barbiturates, opiates, and alcohol may alter a patient's tolerance to anesthetic drugs.
  - 2. "Tranquilizing drugs" - Preoperative use of these drugs reduces the required dosage of general anesthetics. The three important groups are as follows:
    - a. Meprobamates - These have little effect on the conduct of anesthesia.
    - b. Rauwolfia derivatives (reserpine, etc.) - Use of these drugs predisposes to severe hypotension during anesthesia even though administration has been terminated 1-2 weeks before anesthesia. This hypotension responds to vasopressors, but larger than usual dosages must be given.
    - c. Phenothiazine derivatives (chlorpromazine, etc.) - Use of these drugs predisposes to hypotension during anes-

thesia which may not respond even to increased doses of
vasopressor drugs.
3. Corticoids - Prolonged use of cortisone (even though discon-
tinued 1 month or more prior to anesthesia) leads to hypo-
function of the adrenal cortex, which impairs the physiologic
responses to the stress of anesthesia and surgery. Such a
patient should receive cortisone immediately before, during,
and after surgery.
4. Any drugs which may have to be continued during anesthesia
or which may have a bearing on prognosis and complications
(e. g., insulin, glyceryl trinitrate, digitalis).
C. History of Allergies: Asthma and drug sensitivities play an
important role in the choice of agents used.
D. Presence of Food in Stomach: Note the interval since food was
last taken. Pain and anxiety may delay the emptying time of
the stomach. Regurgitation and aspiration are the most seri-
ous complications of general anesthesia.

Physical Examination.
The preanesthetic physical examination should be complete;
special emphasis should be placed on the following:
A. Mental and emotional status.
B. Cardiac status (e. g., decompensation, cyanosis, edema).
C. Status of the respiratory system, with particular reference to
(1) obstruction of the airway; (2) conditions which may lead to
obstruction of the airway (foreign bodies, such as dentures);
and (3) other disorders which may hinder gas exchange, such
as pneumothorax, emphysema, poliomyelitis, and abdominal
distention.
D. Physical deformities or abnormalities which complicate or con-
traindicate specific technics. For example, the patient should
be examined for spinal or sacral deformities if spinal or caudal
anesthesia is contemplated, and ankylosis and other mandibular
and oral pathology may render tracheal intubation difficult.

Special Studies.
The minimum preanesthetic laboratory evaluation of a patient
scheduled for surgical anesthesia should include a complete uri-
nalysis and Hgb. or Hct. Individual cases may require chest x-
rays, blood volume and serum electrolyte determinations, Ecg.'s,
and other laboratory tests aimed at the diagnosis or evaluation of
disorders which must be corrected before anesthesia can proceed
safely.

PREPARATION OF THE PATIENT FOR ANESTHESIA

Medical Preparation.
The patient scheduled for surgical anesthesia should be in the
best physical condition possible within the limits of medical treat-
ment and the urgency of the surgery. The following conditions must
be treated prior to anesthesia in all except extreme emergencies:
(1) shock, (2) anemia, (3) hypovolemia, (4) electrolyte imbalance,
(5) cardiac decompensation, (6) diabetic acidosis, and (7) acute

inflammation of the respiratory system.

The stomach must be allowed to empty itself or must be emptied by gastric tube or induced vomiting.

Patency of the airway must be assured. Tracheostomy made necessary by severe upper respiratory obstruction should be performed under local anesthesia; general anesthesia must not be induced until the tracheostomy has been done.

Psychologic Preparation.

The anesthetist must gain the patient's confidence and make every effort to allay his fears. A personal relationship should be established. The calm, reassured patient is more easily anesthetized.

Pharmacologic Preparation.

The patient should arrive at the operating room in a drowsy but cooperative state and with minimal respiratory depression.

A. Purposes of Preanesthetic Medication: To ensure a good night's rest before surgery, to diminish fear and anxiety and to produce a mild euphoria, to decrease metabolism and anesthetic requirements, to produce amnesia, to decrease secretions in the mouth and respiratory tract, to decrease autonomic reflexes (e.g., cardiac irregularities), to counteract toxic manifestations of local anesthetics, and to minimize postanesthetic nausea and vomiting.

Common Drugs and Average Doses of Drugs for Premedication (Adults)*

Drug	Dosage	Route and Time
Barbiturates		
Pentobarbital (Nembutal®)	50-200 mg. ($3/4$-3 gr.)	Give orally $1^1/2$-2 hours before induction;
Secobarbital (Seconal®)	50-200 mg. ($3/4$-3 gr.)	or I.M. 30 minutes before induction;
Amobarbital (Amytal®)	50-200 mg. ($3/4$-3 gr.)	or I.V. 15 minutes before induction.
Vinbarbital (Delvinal®)	100-200 mg. ($1^1/2$-3 gr.)	
Narcotics		
Morphine sulfate	5-15 mg. ($1/12$-$1/4$ gr.)	Give subcut. 1 hour before induction; or
Meperidine (Demerol®)	50-150 mg. ($3/4$-$2^1/2$ gr.)	I.M. 45 minutes before induction; or
Alphaprodine (Nisentil®)	30-60 mg. ($1/2$-1 gr.)	I.V. 15 minutes before induction.
Belladonna Alkaloids		
Atropine	0.2-0.6 mg. ($1/300$-$1/100$ gr.)	Give subcut. with the narcotic before induction.
Scopolamine	0.2-0.6 mg. ($1/300$-$1/100$ gr.)	

*Dosage of barbiturates and narcotics should be reduced when premedication includes tranquilizers.

PEDIATRIC PREMEDICATION* COMPARABLE DOSES OF NARCOTICS

Age	Average Weight (lb.)	Pentobarbital (Nembutal²) or Secobarbital (Seconal²) mg. (gr)	Atropine or Scopolamine mg. (gr.)	Morphine mg (gr)	Meperidine (Demerol²) mg. (gr)	Alphaprodine (Nisentil³) mg. (gr.)
Newborn	7	-	0 1 (1/600)	-	-	-
6 months	16	30 (1/2)	0.2 (1/300)	-	-	-
1 year	21	50 (3/4)	0.2 (1/300)	1 (1/60)	10 (1/6)	4 (1/15)
2 years	27	60 (1)	0.3 (1/200)	1.5 (1/40)	20 (1/3)	8 (1/8)
4 years	35	90 (1 1/2)	0.3 (1/200)	3 (1/20)	30 (1/2)	12 (1/5)
6 years	45	100 (1 1/2)	0.4 (1/150)	4 (1/15)	40 (2/3)	15 (1/4)
8 years	55	120 (2)	0.4 (1/150)	5 (1/12)	50 (3/4)	20 (1/3)
10 years	65	150 (2 1/2)	0.4 (1/150)	6 (1/10)	60 (1)	25 (3/8)
12 years	85	150 (2 1/2)	0.6 (1/100)	8 (1/8)	80 (1 1/4)	30 (1/2)

The above dosage scale is for well developed patients of average weight. Reductions must be made for under-weight or poorly developed patients No barbiturate is given to patients under 6 months of age; no narcotic to patients under 1 year of age Barbiturates are given rectally at least 90 minutes before operation Dissolve barbiturate in 10 ml of water and inject rectally. Morphine and atropine are given subcut 45 minutes before operation

B. Drugs Used for Preanesthetic Medication: (The tables on pp. 67 and 68 list the commonly used preanesthetic drugs and their recommended dosages.)
   1. Barbiturates - For general sedation, relief of anxiety, hypnosis, and for protection against local anesthetic reactions.
   2. Narcotics - For analgesia, euphoria, and general sedation.
   3. Belladonna derivatives (scopolamine, atropine) for drying secretions and other vagolytic effects. Scopolamine also produces sedation and amnesia.
   4. Ataractics - For tranquilizing effect and to diminish nausea (see p. 622).
   5. Miscellaneous (chloral hydrate, paraldehyde) for general sedation.
C. Individualization of Drugs and Dosages:
   1. Age - Infants and children can be premedicated according to the table on p. 68. The very aged require little or no premedication.
   2. Physical and mental condition - These may increase or greatly diminish the suggested doses.
   3. Anesthetic agent - See the table below for examples.

Selection of Premedication for Various Anesthetic Agents*

Anesthetic Agent	Barbiturates	Narcotic	Atropine or Scopolamine
Ether	++	+	+++
Cyclopropane	++	+	++
Thiopental with N_2O	++	+++	++
Regional Anesthesia Low dosage (sub-arachnoid)	++	++	+
Large dosage (epidural, nerve block, infiltration, etc.)	+++	++	+

+ None or reduced dosage. ++ Desirable. +++ Indicated.
*For selection of drugs in infants and children, see p. 68.

FACTORS INFLUENCING THE SELECTION OF ANESTHETIC AGENTS AND TECHNICS

All of the factors outlined below must be taken into consideration in deciding what type of anesthesia will be used. With few exceptions (e.g., inability to use ether in open chest surgery with cautery), no single factor excludes the consideration of the others.

Patient Factors.
  A. Patient's preferences and prejudices.
  B. Patient's condition.

AGENTS USED FOR GENERAL ANESTHESIA

	Diethyl Ether $(C_2H_5)_2O$	Chloroform $CHCl_3$	Divinyl Ether (Vinethene) $(C_2H_3)_2O$	Ethyl Chloride C_2H_5Cl	Trichloroethylene (Trilene) C_2HCl_3
Concentration in Inspired Mixture For:					
Analgesia	<1%	0.25-0.75%	0.2%	2-3%	0.5-1.5%
Anesthesia	3.5-4.5%	0.75-1.65%	2-4%	3.6-4.5%	5-7.5%
Respiratory Arrest	6.7-8%	2%	10-15%	6%	
Use With Soda Lime	Yes.	Yes.	Yes.	Not recommended.	DANGEROUS.
Depth and Use	Any depth required for surgery.	Rarely used. Light anesthesia.	Light anesthesia for short procedures.	Not recommended.	Analgesia only.
Cardiac Effects					
Arrhythmias	Occasional.	Frequent.	Occasional.	Frequent.	Frequent.
Myocardium	Depressed.	Markedly depressed.	Depressed.	Depressed.	Depressed.
Use with epinephrine or norepinephrine	Yes.	DANGEROUS.	Yes.	DANGEROUS.	DANGEROUS.
Respiratory Effects					
Irritating to respiratory passages	Yes.	Not in recommended concentrations.	Yes.	No.	No.
Secretions	Increased.	Slightly increased.	Increased.	Less than with ether.	Slightly increased.
Respiration	Stimulated, then depressed.	Depressed. Circulation may fail before respiration.	Stimulated, then depressed.	Hyperpnea in light anesthesia. Circulation may fail before respiration.	Tachypnea with increasing depth.
Liver Toxicity	Depression of function.	Marked.	Prolonged anesthesia causes central necrosis. Less toxic than chloroform.	Yes.	Yes.
Muscle Relaxation	Excellent.	Excellent.	Not used for relaxation.	Depth for relaxation dangerous.	Poor.
Explosive	Yes.	No.	Yes.	Yes.	Not in anesthetic concentrations.
Recovery	Slow. Nausea and vomiting frequent.	More rapid than ether. Nausea and vomiting common.	Rapid. Nausea and vomiting uncommon.	Rapid. Nausea and vomiting frequent.	Rapid. Nausea and vomiting less than with ether.

	Halothane (Fluothane) $CF_3CHBrCl$	Nitrous Oxide* N_2O	Ethylene* C_2H_4	Cyclopropane C_3H_6
Concentration in Inspired Mixture For:				
Analgesia	< 0.5%	20-40%	20-25%	3-5%
Anesthesia	0.4-1.6%	85-90%	80-90%	5-23%
Respiratory Arrest				23-40%
Use With Soda Lime	Yes.	Yes.	Yes.	Yes.
Depth and Use	Light to moderate depth anesthesia.	1st plane anesthesia.	1st plane anesthesia.	Moderate depth.
Cardiac Effects				
Arrhythmias	Infrequent; bradycardia.	Rare.	Rare.	Frequent.
Myocardium	Depressed; hypotension.	Slight depression.	Slight depression.	Moderate depression.
Use with epinephrine or norepinephrine	Heart sensitized to pressor amines.	Yes.	Yes.	DANGEROUS. May produce ventricular fibrillation.
Respiratory Effects				
Irritating to respiratory passages	No.	No.	No.	Non-irritating with < 50%.
Secretions	Not increased.	Not increased.	Not increased.	Minimal.
Respiration	Depressed; tachypnea.	Normal.	Normal.	Depressed.
Liver Toxicity	Not determined.	No.	No effect.	Not significantly depressed.
Muscle Relaxation	Good.	No relaxation.	Poor.	Good.
Explosive	No.	No, but supports combustion.	Yes.	Yes.
Recovery *	Moderately rapid. Nausea and vomiting infrequent.	Rapid. Nausea and vomiting uncommon.	Rapid. Nausea and vomiting less than with ether.	Rapid. Nausea and vomiting frequent.

*Nitrous oxide and ethylene should not be administered with less than 20% oxygen. The real danger in their use is from hypoxia and not from the gases themselves.

Surgical Requirements.
 A. Speed and skill of the surgeon; individual skill and experience
    of the anesthetist with the agents and equipment at his disposal.
 B. Site of surgery and position of patient.
 C. Use of electrocautery.
 D. Need for muscle relaxation.
 E. Open chest.

TYPES OF GENERAL ANESTHESIA

General anesthesia is a state of drug-induced CNS depression
characterized by (1) analgesia and amnesia; (2) unconsciousness,
and (3) loss of reflexes and muscle tone. Drugs producing this
state must cause no permanent tissue change; must be reversible
in action by excretion or destruction; and must have minimal side
effects on other tissues.

Different methods of administration all serve the same purpose,
i.e., to introduce the agent into the blood stream for transport to
the CNS. The route of administration (inhalation, gastrointestinal
absorption, or injection into veins or tissues) depends upon the
physical properties of the agent.

Inhalation Anesthesia. (See table on pp. 70-71.)
Inhalation anesthesia is used to administer gases (cyclopropane,
nitrous oxide, and ethylene) and volatile liquids (ethers, chloroform,
trichloroethylene, halothane, ethyl chloride).
 A. Technics:
    1. Open drop - This is the simplest and oldest method and re-
       quires the least equipment. A volatile liquid (ethers, chloro-
       form) is administered drop by drop onto the gauze or cloth
       covering of a wire frame mask applied over the mouth and
       nose. The inhaled concentration is controlled by the rate of
       drip. Induction is accomplished with a slow rate of drip,
       and the rate is increased to the patient's tolerance. When
       the desired depth of anesthesia is reached, the rate of ad-
       ministration is slowed to stabilize the physical signs of that
       depth.
    2. Insufflation - This method consists of blowing anesthetic
       vapors or gases into the mouth, pharynx, or trachea. Some
       type of apparatus or anesthesia machine is required for
       metering gas flows and vaporizing volatile agents. Insuf-
       flation has its greatest usefulness in operations for which a
       mask cannot be used (e.g., tonsillectomy).
    3. Nonrebreathing - As the term indicates, nonrebreathing
       anesthesia supplies a continuous fresh quantity of anesthetic
       agent with adequate oxygen through an apparatus which evac-
       uates each exhalation to the atmosphere. Leigh valves and
       Ayre's T pieces are examples of these apparatus.
    4. Partial and total rebreathing - These methods require an-
       esthesia machines and are named according to the amount
       of exhalation the patient is required to rebreathe. A means
       of absorbing the exhaled CO_2 must be provided in all in-
       stances except where the rebreathing is minimal.

B. Anesthesia Machines: Anesthesia machines make possible:
  1. The use of anesthetic gases and oxygen supplied in compressed form in tanks.
  2. Accurate metering of these gases.
  3. The vaporization of volatile liquids.
  4. The absorption of CO_2.
  5. The conservation of expensive agents.
  6. Artificial respiration (by using the reservoir bag as a bellows for inflation of the lungs).

Intravenous Anesthesia.
  A. Commonly Used Agents:
    1. Barbiturates - Thiopental (Pentothal®) or thiamylal (Surital®) is administered slowly in 2.5% solution until loss of consciousness occurs. Dosage is limited by respiratory depression. Increments may be added as needed.
    2. Narcotics.
  B. Advantages: Ease of administration with a rapid and pleasant induction
  C. Disadvantages: Lack of control after administration. The duration of action depends upon the destruction or renal excretion of the agent.

Rectal Anesthesia.
  A. Commonly Used Agents· Barbiturates, tribromoethanol (Avertin®).
  B. Advantages: Ease of administration with pleasant induction when the intravenous approach is impractical (children, obese patients).
  C. Disadvantages: Same as for intravenous anesthesia; in addition, the rate of absorption is slow and the amount of anesthetic which can be absorbed in this way is difficult to predict.

Balanced Anesthesia.
  In balanced anesthesia several agents are administered by 1 or more of the above methods. Each agent is used for its most desirable properties, and none is given in toxic amounts. By variations in the dose of different agents the anesthetic is made to suit the surgical requirement. A typical example is the combination of (1) an intravenous barbiturate for its hypnotic effect, (2) an intravenous narcotic for analgesia, (3) nitrous oxide by inhalation for maintenance of light anesthesia, and (4) muscle relaxants to diminish or abolish muscle tone.

Muscle Relaxants.
  Curare and allied drugs are listed in the table on p. 624. These agents act peripherally at the myoneural junction to diminish or abolish the action of the skeletal muscle. In sufficient doses they paralyze all voluntary muscles, including those of respiration. **Caution:** Curare and allied drugs must never be used unless a means of administering oxygen under positive pressure is immediately available. Following the administration of these agents the patient must be observed until all effects have worn off.

STAGES AND PLANES OF ANESTHESIA

The 4 small black rectangles represent zones, as follows:

1. Conjunctival column - Disappearance and reappearance of lid reflex.
2. Pharyngeal column - Appearance of swallowing (upper border of plane i) and of vomiting (lower border of Stage II).
3. Laryngeal column - Disappearance of carinal reflex.

The large plus and minus signs refer to the presence or absence of the indicated reflex.

Respiration Column.

Thoracic and abdominal inspiration is shown moving away from the mid-line; expiration, toward the mid-line. Regularity, rate, and depth are shown for each stage and plane, in comparison with the normal.

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STAGES OF ANESTHESIA	RESPIRATION Thoracic	RESPIRATION Abdominal	PUPIL SIZE NO MEDICATION	PUPIL SIZE MORPHINE 15 mg. AND ATROPINE 0.4 mg.	PUPIL SIZE MORPHINE 15 mg.	EYE-BALL ACTIVITY	REFLEXES CORNEAL	REFLEXES CONJUNCTIVAL	REFLEXES PHARYNGEAL	REFLEXES LARYNGEAL	REFLEXES CUTANEOUS	REFLEXES PERITONEAL	SOMATIC MUSCLES
I ANALGESIA			◉	◉	⊙	VOLUNTARY	+	+	+	+	+	+	NORMAL TONE
II DELIRIUM			◉	◉	◉	+++ +++ +++ +	+	+	▮ +	+	+	+	UNINHIBITED ACTIVITY
III PLANE i (SURGICAL)			⊙	◉	⊙	FIXED	+ −	▮ −	+ −	+ −	+ −	+ −	RELAXATION • SLIGHT
III PLANE ii (SURGICAL)			◉	◉	⊙	FIXED	−	−	−	−	−	+ −	• MODERATE
III PLANE iii (SURGICAL)			◉	◉	◉	FIXED	−	−	−	▮	−	−	• MARKED
III PLANE iv (SURGICAL)			◉	◉	⊙	FIXED	−	−	−	−	−	−	• MARKED
IV MEDULLARY PARALYSIS				◉	◉		−	−	−	−	−	−	• EXTREME

MANAGEMENT OF GENERAL ANESTHESIA

Regulation of Depth of Anesthesia.

By ''depth of anesthesia'' is meant the degree of depression produced by the anesthetic agent. Increasing doses of anesthetic agents progressively depress physiologic functions such as respiration, cardiac action, muscle tone, and reflexes. Changes in these functions are the signs of anesthesia. Different agents depress these functions in a different order and to different degrees. For example, at the same level of respiratory depression, the muscle relaxant and circulatory effects of ether, chloroform, cyclopropane, and halothane are very different. The clinical signs at various stages and planes of ether anesthesia were classically described by Guedel (see p. 74). All of these signs are useful in monitoring a patient's response to any agent, but their significance in determining the depth of anesthesia will vary with the agent. By observing these signs one strives to provide satisfactory surgical conditions with minimal depression of those functions which are vital.

Care of the Unconscious Patient.

The nervous system depression produced during anesthesia permits painless surgery but deprives the patient of important protective reflexes and homeostatic responses which maintain the efficiency of the cardiorespiratory functions. The anesthetist must correct or compensate for these undesirable effects. The most serious are those which interfere with the adequate oxygen supply to the tissues, i.e., hypoxia. Prevention of hypoxia during anesthesia is the most important task of the anesthetist.

Hypoxia During Anesthesia.

Hypoxia during anesthesia may be due to inadequate pulmonary ventilation or circulatory deficiencies.

A. Inadequate Pulmonary Ventilation:
1. Signs -
   a. Early - Cyanosis and tachycardia.
   b. Late - Bradycardia, failing circulation, dilated pupils, and cardiac arrest.
2. Etiology -
   a. Depression of the respiratory center by narcotics or anesthetics (may affect rate or depth of respirations, or both).
   b. Weakness or paralysis of the muscles of respiration during deep anesthesia or with the use of muscle relaxants.
   c. Obstruction of the respiratory passages - Obstruction is the most frequent of all causes of hypoxia during general anesthesia. It is detected by noticing the signs of obstruction: (1) noisy respiration; (2) increased muscular effort during inspiration; (3) indrawing of the soft tissues of the thorax, such as the suprasternal notch, the supraclavicular fossae, and the intercostal spaces; and (4) a diminished gas movement for the effort involved. This movement can be assessed by observation of the rebreathing bag of the anesthesia machine or by listening with the ear close to the patient's mouth.

Obstruction of the airway may occur anywhere along the tract from the nose and lips to the pleurae (pneumothorax). Any narrowing or occlusion of the air passages or impediments to expansion of the lungs constitutes a form of obstruction. Hundreds of causes have been reported, including foreign bodies, inflammatory disorders, and neoplasms. The 3 most common causes of obstruction are as follows:

(1) Relaxation of the mandibular and lingual muscles, so that the tongue blocks the pharynx.

(2) Laryngospasm - This consists of the reflex closure of the glottis when stimulated during light anesthesia. Thiopental, which does not suppress the laryngeal reflex, predisposes to a severe and obstinate type of laryngospasm.

(3) Vomitus in the throat or trachea.

3. Treatment -

   a. Respiratory depression - Lighten anesthesia and assist respirations.

   b. Muscle weakness or paralysis - Assist or control respirations until muscle power returns.

   c. Obstruction of respiratory passages -

      (1) Relaxation of mandibular and lingual muscles - This is treated by pulling the mandible forward and/or inserting an oropharyngeal airway   The airway must reach behind the tongue and not push the tongue farther back.

      (2) Laryngospasm - Removal of the stimulus, deepening the anesthetic level if possible, or the use of some form of positive pressure to assist respiration, may relieve the spasm. Succinylcholine, 10-20 mg. ($^1/6$-$^1/3$ gr.) I.V. or I.M., may be resorted to if other methods fail.

      (3) Vomiting - This is best treated prophylactically by emptying the stomach before general anesthesia. Aspirated vomitus must be suctioned out through a bronchoscope.

      (4) Tracheal intubation (see below) must be resorted to if treatment by the above measures is not successful.

B. Circulatory Deficiencies:

   1. Signs - Falling BP, weak pulse, ashen gray color.

   2. Etiology -

      a. Diminished cardiac output brought on by -

         (1) Myocardial depression by anesthetic drugs (e.g., halothane) or by hypoxia.

         (2) Poor venous return caused by incorrect posture, loss of peripheral venous tone, increased intrathoracic pressure, manipulation of major vessels during surgery, etc.

      b. Diminished circulating blood volume or hemorrhage.

      c. Diminished peripheral arteriolar resistance, most often caused by the depressant actions of many anesthetic drugs on the sympathetic nervous system.

   3. Treatment -

      a. Reduction in depth of anesthesia. Be certain that

oxygenation is adequate.
 b. Replacement of blood volume.
 c. Change of position (Trendelenburg) to aid venous return.
 d. Vasopressor drugs - Epinephrine, ephedrine, or other
    vasopressor drugs should be used as adjuncts to other
    methods of treatment as indicated.

Tracheal Intubation.
 Obstruction of the upper respiratory passages may best be
treated or prevented by inserting an endotracheal tube. This is
done by visualizing the larynx with a laryngoscope and inserting the
tube deep into the trachea. Muscle relaxant drugs or deep ether
anesthesia may be used to facilitate nontraumatic intubation. The
tube should not reach the carina (teeth to carina in adults is 24-26
cm.), and must be narrow enough to enter the glottis with ease.
 Intubation is indicated (1) in upper respiratory obstruction, (2)
to remove the anesthetic mask from the vicinity of the surgical
field, (3) to facilitate controlled respiration (e.g., open chest op-
erations), and (4) prophylactically, when emergency intubation
would be difficult during surgery (e.g., when the face-down posi-
tion is necessary).

REGIONAL ANESTHESIA
(Local Anesthesia)

 Regional anesthesia is used to render a selected part of the
body insensitive to pain and, if necessary, incapable of muscular
action; the patient retains consciousness. Regional anesthesia is
achieved by blocking the conduction of nerve impulses from and to
a well-defined area. The interruption of the nerve impulses may
be done anywhere along the length of the peripheral nerves from
their motor or sensory endings to their entry into the CNS in the
spinal canal or skull.

Agents Used in Regional Anesthesia.
 Drugs used for regional anesthesia must have a limited duration
of action and must not cause permanent damage to the nerves blocked
or to other tissues. Their therapeutic dose must be well below the
toxic dose.
 The table on p. 78 lists the local anesthetic drugs commonly
employed together with their uses, individual properties, dosages,
and toxic amounts.

Toxic Manifestations of Local Anesthetics.
 Local tissue toxicity is rare.
 Systemic reactions are more common than local reactions and
may affect the CNS or the cardiovascular system, or may be of an
allergic nature. Reactions occur when toxic concentrations are ab-
sorbed from the site of (topical) application or injection. The fac-
tors which favor such concentrations are (1) accidental intravascular
injections and (2) rate of absorption. Rate of absorption is increased
by large total dose or by vascularity at the site of injection (reduced
by the use of epinephrine).

DRUGS USED FOR LOCAL ANESTHESIA

	Cocaine	Procaine (Novocaine®, Novocaine®)	Tetracaine (Pontocaine®)	Lidocaine (Xylocaine®)	Dibucaine (Nupercaine®)	Piperocaine (Metycaine®)
Potency*	3	1	10	1.5-2	15	1.4
Toxicity*	4	1	10	1-1.5	10-15	1.4
Stability	Cannot be auto-claved.	Stable.	Stable.	Stable.	Stable.	Stable.
Recommended Concentration for Anesthesia:						
Caudal-epidural	Do not use.	2%	0.2%	1.2-2%	Not commonly used.	1.0-1.5%
Nerve blocks	Do not use.	1-2%	0.1-0.2%	1-2%	Not commonly used.	1.0-1.5%
Infiltration	Do not use.	0.25-0.5%	0.05-0.1%	0.5%	Not commonly used.	0.5-1%
Topical						
Eye	1%	Not effective.	0.5%	0.5%	0.1%	2%
Nose and throat	4-10%	Not effective.	2%	1-2%	0.5-2%	2-10%
Urethra	Absorption rapid.	Not effective.	Too toxic.	1%	0.05-0.2%	1-4%
Onset of Action	Immediate	5-15 minutes.	10-20 minutes.	5-10 minutes	8-15 minutes.	5-10 minutes.
Duration of Action	30-60 minutes.	45-60 minutes.	1½-3 hours.	1-2 hours	2-3 hours.	1 hour.
Total Maximum Dose†	100-200 mg.	1 Gm.	50-100 mg.	500 mg.	35 mg.	1 Gm.
Spinal Anesthesia:						
Recommended Conc.	Do not use.	3-5%	0.3-0.5%	Not commonly used	0.06%‡ 0.25%§	3-5%
Dose Range		50-200 mg.	5-20 mg.		4-13 mg.‡ 2.5-7.5 mg.§	50-125 mg.
Onset of Action		3-5 minutes.	5-10 minutes.		7-15 minutes.	3-5 minutes.
Duration of Action		45-60 minutes.	1½-2 hours.		2½-3 hours.	1-1½ hours.

*Potency and toxicity relative to procaine = 1.   ‡Hypobaric.
†Maximum dose administered at one time   §Hyperbaric.

1. Duration of anesthesia can be prolonged by using epinephrine or phenylephrine (Neo-Synephrine®).
   (a) For epidural and nerve blocks, use epinephrine with resultant concentration of 1:200,000.
   (b) For spinal anesthesia use epinephrine, 0.1-0.3 mg., or phenylephrine (Neo-Synephrine®), 2-4 mg.
2. AUTOCLAVE all drugs used for epidural or spinal anesthesia. DO NOT COLD STERILIZE.
3. Solutions for spinal anesthesia are made hyperbaric by using 10% dextrose as the diluent.

A. CNS Reactions: Twitches and tremors, first seen about the mouth and eyes, progress to convulsions which so impair the coordination of respiratory efforts that severe hypoxia ensues. The prognosis depends upon the severity and duration of hypoxia and the physical condition of the patient. Varying degrees of permanent brain damage may occur, or the patient may die in convulsions.

   Convulsions are treated with oxygen therapy and intravenous barbiturates. Oxygen therapy is the more important and takes precedence. Prevent hypoxia during operation by administering oxygen. If convulsions develop, inject barbiturates intravenously. **Caution:** Small doses (100 mg. of thiopental, pentobarbital, or amobarbital) are adequate and more should not be given; large doses may synergize with postconvulsive depression and lead to severe central respiratory depression.

B. Cardiovascular Reactions: Tachycardia, palpitation, and anxiety are more likely to be caused by the epinephrine which is added to the local anesthetic.

   Anaphylactic shock (see p. 13) is a rare complication of regional anesthesia and requires vigorous antishock measures. The sudden cardiovascular collapse with absent BP and pulse and ashy pallor can only be treated by cardiac massage (see Cardiac Arrest, p. 39).

C Allergic manifestations in susceptible individuals include the usual variety of skin manifestations and bronchospasm. Treatment is with epinephrine or antihistamines.

Technics of Regional Anesthesia.

Surgical asepsis must be maintained. Do not inject into an infected area   Always aspirate before injecting to avoid intravascular injection.

A. Topical: Application of readily absorbed local anesthetic agents (cocaine, tetracaine) on mucous surfaces. Examples:
   1. Eye - 4% cocaine.
   2. Pharynx and trachea for bronchoscopy - 4% cocaine, 2% tetracaine (Pontocaine®), or 2% lidocaine (Xylocaine®).
   3. Urethral for cystoscopy - Females: Cocaine, 10%, on cotton swab.   Males: Lidocaine (Xylocaine®), 1%, 10 ml.

B. Infiltration: Injection of local anesthetic into tissues, e. g. , into the fracture site for Colles' fracture, around a laceration to be sutured, around donor areas before taking skin grafts, etc.

C. Field Block: Injection of local anesthetic into tissues surrounding the area to be made insensitive without infiltrating the area itself, e. g. , for excision of sebaceous cysts or lipomas and for herniorrhaphies.

D. Nerve Blocks: Injection of anesthetic agent to permeate nerves at some point proximal to the area to be anesthetized. Almost every peripheral nerve in the body may be blocked if the anatomy of the region is known. In many cases, eliciting paresthesia with the injecting needle helps locate the nerve to be blocked. The drawings on pp. 80-81 illustrate some common nerve blocks useful in outpatient practice.

E. Spinal Anesthesia: In spinal anesthesia the spinal nerves are blocked between their emergence from the cord and their exit

Flexor carpi radialis longus T.
Median nerve
Palmaris longus T.
Ulnar

Radial nerve

Dorsal branch of ulnar nerve
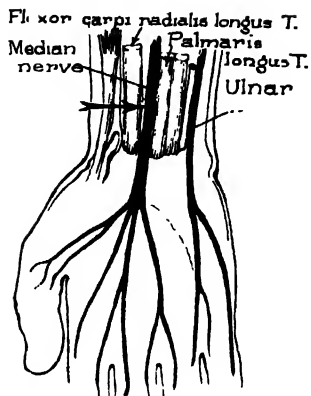
Median Nerve Block at Wrist
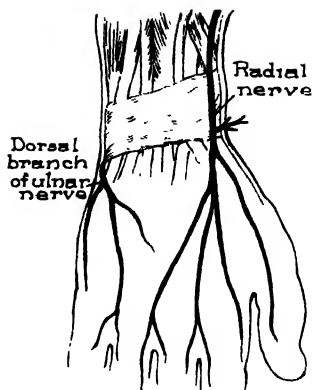
Radial Nerve Block at Wrist
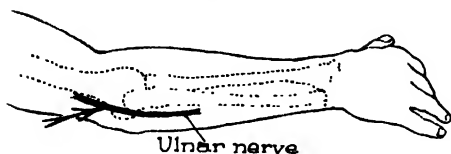
Ulnar nerve

Ulnar Nerve Block at Elbow

Brachial Plexus Block in Axilla

Pectoralis major
Median nerve
Musculocutaneous n.
Radial nerve
Ulnar nerve
Med brach cut n.
Med antibrach.cut.n.
Teres major and Latissimus dorsi muscles
Triceps
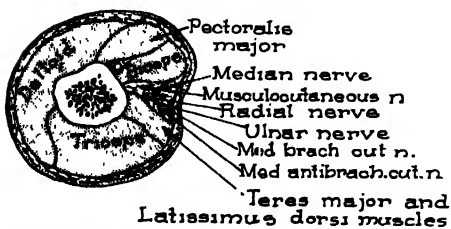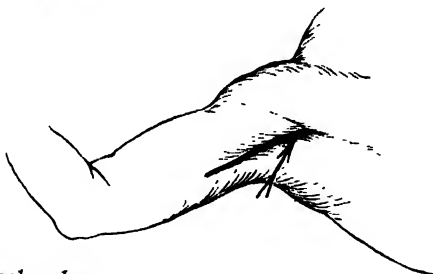
Cross-section of Arm at Level of Brachial Plexus

(Heavy arrows show point of injection of anesthetic agent.)
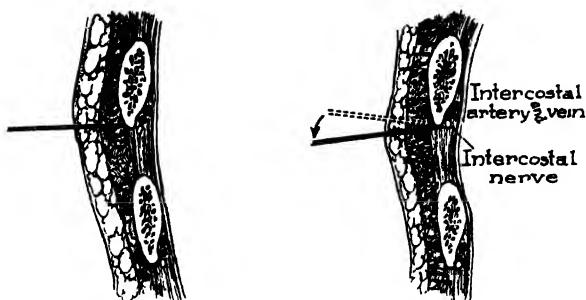
Intercostal Nerve Block. Left: Needle locating lower edge of
rib. Right: Needle under edge of rib for injection of anesthetic
agent.
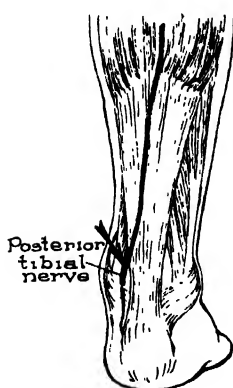
**Posterior Tibial Nerve
Block at Ankle**

**Anterior Tibial Nerve
Block at Ankle**
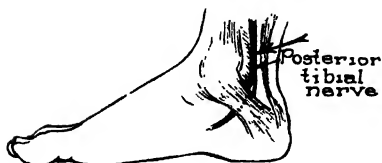
**Posterior Tibial Nerve
Block at Ankle.
Side view.**

(Heavy arrows show point of injection of anesthetic agent.)

from the spinal canal through the intervertebral foramens.
This may be done in the subarachnoid space by injecting a local
anesthetic agent into the CSF or into the epidural space at any
level of the vertebral column, including the sacral hiatus. The
table on p. 78 gives the doses of drugs used for spinal anes-
thesia.

1. Types of spinal anesthesia -
   a. Subarachnoid spinal anesthesia - Sterile technic is used;
      ampuls must be autoclaved, not kept in alcohol or other
      disinfectants. With the patient in either the lateral de-
      cubitus or sitting position, lumbar puncture is done at
      any level between the second and fifth lumbar vertebrae.
      When a free flow of CSF is obtained, the anesthetic is in-
      jected into the subarachnoid space. This blocks all spinal
      nerves below the site of injection as well as those above
      the site of injection which are reached by the anesthetic
      solution in its upward spread. This upward spread can
      be controlled by limiting the volume and concentration
      of the injection and by judicious use of posture and the
      curves of the spinal canal to ''float'' or ''sink'' an anes-
      thetic solution which is heavier (hyperbaric) or lighter
      (hypobaric) than the CSF. Dilution by the CSF also limits
      the effective spread. After 20 minutes the solution is
      ''fixed'' and no further spread occurs.
         A ''high spinal'' (reaching the upper thoracic nerves)
      is obtained by injecting a large volume of a hypobaric
      solution with the patient sitting or with a hyperbaric so-
      lution with the patient placed in 5-10° degrees of Tren-
      delenburg.
         Duration of subarachnoid spinal anesthesia depends
      upon the agent selected:

Procaine	-	30-75 minutes
Piperocaine (Metycaine®)	-	45-90 minutes
Tetracaine (Pontocaine®)	-	1-2 hours
Dibucaine (Nupercaine®)	-	2-3 hours

      Increased dosages increase the duration of anesthesia
      only slightly. The addition of 0.2-0.4 ml. of 1:1000 epi-
      nephrine or 3 mg. of phenylephrine (Neo-Synephrine®)
      increases the duration of anesthetic effect 30-50%.
   b. Epidural spinal anesthesia - A needle is introduced into
      the epidural space, either in the lumbar area or into
      the sacral canal, through the sacral hiatus. In the
      lumbar area the space is recognized by advancing the
      needle in the same manner as for a lumbar puncture, but
      stopping when resistance to injection is no longer felt
      even though no CSF can be aspirated. A test dose of local
      anesthetic sufficient to achieve subarachnoid spinal anes-
      thesia (e.g., 5 ml. of 1% lidocaine) but insufficient for
      epidural anesthesia proves in 5 minutes that the sub-
      arachnoid space has not been entered. The anesthetic
      dose is then given either by single injection or through a
      plastic catheter advanced through the needle for continuous

epidural anesthesia. (Caution: Catheters should never be withdrawn through the needle for fear of cutting the catheter in the spinal canal.)

The concentrations used and the rates of onset of the block are the same as in peripheral blocks of large nerves. Lidocaine (Xylocaine®), 1-2%, or tetracaine (Pontocaine®), 0.1-0.2%, may be used. Since the epidural space extends from the foramen magnum to the sacral hiatus, the area anesthetized is limited only by the spread of the injected solution and is subject to considerable individual variation. The dangers of toxicity must be borne in mind because the total dose required may be quite large.

(1) Caudal approach - 20 ml. for sacral nerves for perineal surgery; 30-35 ml. for lumbar and lower thoracic nerves.

(2) Lumbar approach - 20-25 ml. for lower abdominal surgery; 25-30 ml. for upper abdominal surgery.

(3) Repeat doses by catheter require half the initial dose.

2. Comparison of subarachnoid and epidural block -
   a. Advantages of subarachnoid block -
      (1) Technically easier.
      (2) Rapid onset.
      (3) Small drug doses with less chance of toxic reactions.
   b. Advantages of epidural block -
      (1) Fewer postoperative complications, such as spinal headaches.
      (2) Less patient prejudice against this technic.

3. Effects of spinal anesthesia -
   a. Analgesia over distribution of the spinal nerves anesthetized
   b. Paralysis of muscles innervated by the spinal nerves anesthetized. When the block reaches the thoracic nerves, respiration is impaired to the extent that the intercostal muscles are paralyzed; when it reaches the fourth cervical segment, respiration ceases (phrenic nerve paralysis).
   c. Sympathetic paralysis (thoracolumbar outflow from T1 to L2) causes the following:
      (1) Loss of vasomotor tone in the affected area, with peripheral vasodilatation, diminished vascular resistance, and loss of ability to control blood distribution. This impairment leads to a fall in BP which is particularly severe in hypertensive and in hypovolemic states.
      (2) Active intestinal peristalsis.
      (3) Warmth and dryness of skin, which is easily distinguished from the damp pallor seen in shock.

4. General management of patients during spinal anesthesia -
   a. Prevent hypoxia by the administration of oxygen or assisted respiration.
   b. Support circulation by -
      (1) Positioning patient to promote venous return.
      (2) Use of vasopressors (see p. 623). When high spinal

anesthesia is planned, an intravenous infusion should be started before the spinal anesthetic for easy administration of vasopressors. Vasopressors may be given prophylactically before the spinal.

5. Sequelae of spinal anesthesia -
   a. "Spinal headache" - Occurs during the first week after subarachnoid block and may last several days. Treatment consists of keeping the patient flat in bed for the first 24 hours to diminish CSF loss at the site of the puncture of the dura. Incidence is 1-15%, depending upon the size of needle used for the lumbar puncture.
   b. Nerve injuries and transverse myelitis may cause transient or permanent disabilities ranging from minor paresthesias to paraplegia. These sequelae are rare, but their tragic impact gives them notoriety out of proportion to their frequency. They may be due to contaminants, drug sensitivities, or excessive dosages. Cases have been reported following epidural as well as subarachnoid spinal anesthesia.

OXYGEN THERAPY

Oxygen therapy consists of increasing the oxygen concentration of inspired air. At sea level the normal 21% oxygen concentration constitutes 159 mm. Hg (partial pressure) of the total 760 mm. Hg atmospheric pressure. Dead space, water vapor, CO_2 diffusing from the blood, and oxygen diffusing into the blood unite to reduce the alveolar oxygen to the 100 mm. Hg partial pressure normally found in the alveoli. This alveolar oxygen pressure is sufficient to saturate hemoglobin with oxygen, but may be reduced by (1) diminished oxygen partial pressure in the inspired air (e.g., high altitudes), or (2) diminished pulmonary ventilation as seen in respiratory depression or obstruction.

The objective of oxygen therapy is to raise the alveolar oxygen partial pressure and arterial oxygen saturation to normal. If ventilation is inadequate it is a temporary life-saving measure, but it must not be used as a substitute for measures necessary to return pulmonary ventilation to normal (i.e., clearing the airway, etc., as on p. 16).

Signs and symptoms of hypoxia in the conscious patient include tachycardia, restlessness, mental confusion, and air hunger in the early stages; and cyanosis, bradycardia, and loss of consciousness when the hypoxia becomes severe.

Methods of Administration.
' All methods require a source of oxygen (usually tanks of compressed oxygen), reducing valves, and flow meters.

A. Oropharyngeal Catheter: By this method the oxygen is administered through a catheter introduced through the nose and advanced until the tip can be seen below the uvula. Flows of 6-8 L. of humidified oxygen raise the oxygen in the inspired air to 35-40%.

B. Oxygen Mask: A wide variety of rubber and plastic masks with and without rebreathing bags, or valves to offer positive pres-

sure on expiration, are available. They permit higher oxygen concentrations.

C. Oxygen Tents: In these the patient is not encumbered by masks or catheters. High concentrations are possible, but without expert adjustment and supervision oxygen administration by tent may be most inefficient and useless.

POST-ANESTHESIA CARE

Anesthesia does not terminate with closure of the incision; its termination depends upon the elimination of the anesthetic agent by pulmonary or renal excretion or by metabolic destruction. The duration of the period required for recovery from the effects of the anesthetic varies with the agents and technics used (regional anesthesia not excepted). During this time the protective reflexes and homeostatic responses which control vital respiratory and circulatory functions return gradually. The anesthetist must continue to provide the same care as during surgery until the patient is conscious and his vital signs have stabilized. Above all, **the patient must not be left unattended.** Respiration, circulation, and state of reactivity must be monitored at frequent intervals.

The serious hazards peculiar to the postanesthetic period are respiratory and circulatory collapse.

Respiration.
HYPOXIA IS ONE OF THE MOST FREQUENT CAUSES OF POSTOPERATIVE RESTLESSNESS.

Respiratory depression may be due to the prolonged effect of the agents used during surgery or may be caused by postoperative pain medication. Increasing respiratory depression may occur as the painful stimuli of surgery subside. Respiratory obstruction may be due to undrained secretions, tight dressings or casts, etc. Hematomas, pneumothorax, and atelectasis must be kept in mind.

Treatment is by removal of all impediments to respiration, oxygen therapy, and assisted respiration if necessary.

Circulation.
Postoperative hypotension may be caused by drug depression, blood loss, sudden elimination of excess CO_2, and hypoxia. Two or more of these factors are usually involved.

Treatment consists of placing the patient in the shock position (elevate foot of bed), oxygen therapy, fluid replacement, and (rarely) the use of vasopressor drugs.

4 . . .

Nutrition

Energy for physiologic processes is provided in the metabolism of carbohydrate, fat, and protein. The daily requirement for energy (the daily caloric need) is the sum of the so-called basal energy demand (the energy required merely to maintain life) plus that required for additional activity. Extra energy is needed also during periods of growth, pregnancy, or convalescence. Accelerated metabolism, as in hyperthyroidism or during fever, also increases the energy demand. For example, in fever there is an increase of approximately 12% of the basal caloric requirement per degree centigrade (8% per degree Fahrenheit) rise in temperature over normal.

The total caloric requirements of healthy persons of various ages and sex is given in the table on p. 88. These intakes provide for average activity. However, caloric requirements in pathologic states (even at bed rest) increase sometimes to very high levels, as indicated below.

Daily Caloric Requirements in Adult Patients

Afebrile, minor injury or illness, at bed rest	2000 Cal.
Severe injury or illness	2500-4000 Cal.
Previously depleted	3500 Cal.
Severe chills, fever	5000 Cal.

PROTEIN NUTRITION

Protein is not replaceable by any other food and is therefore absolutely necessary in all diets. The recommended daily intakes of protein for healthy individuals are shown in the table on p. 88. However, the quantity of protein required by various surgical patients is highly variable, ranging from 80 Gm. /day to a high of 200-300 Gm. /day or more in severe burns. At least 100 Gm. of protein should be given daily to sick or injured persons.

The metabolism of protein is profoundly affected by the stress incurred in such pathologic states as infections, injuries, major surgery, and burns. For a variable period after the onset of stress patients undergo a period of obligatory negative nitrogen balance. Prolonged bed rest will also induce a negative nitrogen balance. It is difficult or impossible to avoid this apparently physiologic catabolic response to stress even after greatly increased intakes of protein and calories have been provided.

Depletion of body protein is associated with prolonged convalescence, poor healing of wounds, more frequent and more severe postoperative complications, increased susceptibility to infection, anemia, edema, impaired gastrointestinal motility, and skeletal muscle weakness.

The efficiency with which administered protein is utilized for tissue repair depends upon an adequate intake of calories from non-protein nutrients. In the postoperative period an intake of as much as 0.5 Gm. of nitrogen (1 Gm. of nitrogen = 6.25 Gm. protein) and 45 Calories/Kg. may be necessary to restore positive nitrogen balance in a nutritionally depleted patient.

CARBOHYDRATES AND FATS IN NUTRITION

In the usual well-balanced diet about 50-60% of the total calories may be derived from carbohydrates and 20-30% from fats. Necessary increases in caloric intake are usually achieved by increasing the fat content of the diet.

The presence of carbohydrate is essential to permit the efficient utilization of fats without the development of ketosis. At least 5 Gm. of carbohydrate/100 Calories of the total diet are required for this purpose. Surgical patients maintained exclusively on intravenous fluids will probably obtain much of their required energy from their own reserve stores of fat because of the difficulty of providing adequate nutrition when only the parenteral route is available to supply nutrient substances. Under these circumstances an intake of as little as 100 Gm. of carbohydrate (2000 ml. of 5% dextrose) is adequate to prevent ketosis.

VITAMINS

The vitamin requirements of healthy persons are given in the table on p. 88. Normal persons on an adequate diet can secure all of the required vitamins from natural foods. However, in disease states in which digestion or assimilation is impaired or where the requirements are increased, vitamin supplementation is necessary.

The recommended **therapeutic daily doses** of the most important vitamins are as follows:

Thiamin (B_1)	5-10 mg.
Riboflavin (B_2)	5-10 mg.
Niacinamide	100 mg.
Calcium pantothenate	20 mg.
Pyridoxine (B_6)	2 mg.
Folic acid (folacin)	1.5 mg.
Vitamin B_{12}	4 mcg.
Ascorbic acid (vitamin C)	500 mg.

For short-term vitamin supplementation only thiamin, riboflavin, niacinamide, and ascorbic acid need be given. If prolonged intravenous alimentation is required or if an adequate intake of vitamins cannot be obtained from the diet, all of the above vitamins should be given in the doses listed.

When vitamins are added to solutions to be given intravenously, large losses are likely to occur into the urine. For this reason intramuscular or subcutaneous injection is preferred if the oral route cannot be used.

RECOMMENDED DAILY DIETARY ALLOWANCES† (1)

Designed for the Maintenance of Good Nutrition of Healthy Persons in the U.S.A.

(Allowances are intended for persons normally active in a temperate climate.)

	Age Years	Weight Kg. (lb.)	Height cm. (in.)	Calories	Protein Gm.	Calcium Gm.	Iron mg.	Vitamin A I.U.	Thiamine mg.	Riboflavin mg	Niacin mg.	Ascorbic Acid mg.	Vitamin D I.U.
Men	25	70 (154)	175 (69)	3200§	70	0.8	10	5000	1.6	1.8	21	75	
	45	70 (154)	175 (69)	3000	70	0.8	10	5000	1.5	1.8	20	75	
	65	70 (154)	175 (69)	2550	70	0.8	10	5000	1.3	1.8	18	75	
Women	25	58 (128)	163 (64)	2300	58	0.8	12	5000	1.2	1.5	17	70	
	45	58 (128)	163 (64)	2200	58	0.8	12	5000	1.1	1.5	17	70	
	65	58 (128)	163 (64)	1800	58	0.8	12	5000	1.0	1.5	17	70	
	Pregnant (second half)			+300	+20	1.5	15	6000	1.3	2.0	+3	100	400
	Lactating (850 ml. daily)			+1000	+40	2.0	15	8000	1.7	2.5	+2	150	400
Infants**	0-1/12**				See Footnote								
	2/12-6/12	6 (13)	60 (24)	Kg.×120	**	0.6	5	1500	0.4	0.5	6	30	400
	7/12-12/12	9 (20)	70 (28)	Kg.×100		0.8	7	1500	0.5	0.8	7	30	400
Children	1 - 3	12 (27)	87 (34)	1300	40	1.0	7	2000	0.7	1.0	8	35	400
	4 - 6	18 (40)	109 (43)	1700	50	1.0	8	2500	0.9	1.3	11	50	400
	7 - 9	27 (60)	129 (51)	2100	60	1.0	10	3500	1.1	1.5	14	60	400
	10-12	36 (79)	144 (57)	2500	70	1.2	12	4500	1.3	1.8	17	75	400
Boys	13-15	49 (108)	163 (64)	3100	85	1.4	15	5000	1.6	2.1	21	90	400
	16-19	63 (139)	175 (69)	3600	100	1.4	15	5000	1.8	2.5	25	100	400
Girls	13-15	49 (108)	160 (63)	2600	80	1.3	15	5000	1.3	2.0	17	80	400
	16-19	54 (120)	162 (64)	2400	75	1.3	15	5000	1.2	1.9	16	80	400

†The allowance levels are intended to cover individual variations among most normal persons as they live in the United States under usual environmental stresses. The recommended allowances can be attained with a variety of common foods, providing other nutrients for which human requirements have been less well defined. See original reference for more detailed discussion of allowances and of nutrients not tabulated.

§Caloric allowances apply to individuals usually engaged in moderate physical activity. For office workers or others in sedentary occupations they are excessive. Adjustments must be made for variations in body size, age, physical activity, and environmental temperature.

**See original reference for discussion of infant allowances.

(1)Modified and reproduced, with permission, from Publication 589, National Academy of Sciences–National Research Council, 1958.

Deficiencies of fat-soluble vitamins (vitamins A, D, and K) will occur if fat digestion or absorption is impaired, as in obstructive jaundice, biliary fistula, pancreatic disease, or in any disease state which extensively involves the gastrointestinal tract. For the surgical patient a deficiency of vitamin K (almost exclusively an effect of malabsorption) is the most significant. Because this fat-soluble vitamin is essential to the production of prothrombin by the liver, its lack in the body causes hypoprothrombinemia and thus disrupts normal coagulation mechanisms. Preoperative administration of vitamin K by injection is indicated in such cases to raise the prothrombin level to at least 60-70% of normal. The usual daily dose is 2-5 mg. of Menadione Sodium Bisulfite Injection, U.S.P. (Hykinone®), I.V. or I.M. Failure of prompt response to these conservative doses constitutes evidence of severe hepatic damage, and this must be taken into account in assessing the hemorrhagic risks of surgery.

PARENTERAL NUTRITION

Patients who cannot take food orally must be nourished intravenously. By this method it is possible for a limited period to give most of the essential nutrients required for adequate although not ideal nutrition.

Carbohydrates.

The principal problem in parenteral nutrition is to provide sufficient calories. Under the usual circumstances dextrose is relied upon to provide all of the exogenous calories. However, because 1 L. of 5% dextrose (glucose) provides only 200 Calories (1 Gm. dextrose = 4 Calories) and because 3 L./day I.V. is probably an average maximum intake for an adult patient, 5% dextrose solution can provide only about 600 Calories/day. When it is recalled that 2000 Calories/day is the minimum requirement for a bed patient, it becomes obvious that a caloric deficit must often occur when the usual parenteral regimen is employed.

More concentrated solutions of dextrose may be used in an effort to provide more calories, but these solutions are hypertonic and lead to phlebitis. Furthermore, there are limits to the rate at which dextrose can be given intravenously without causing glycosuria, which is wasteful of calories and contributes to dehydration via the polyuria and electrolyte diuresis with which glycosuria is always associated. In general, dextrose should not be given intravenously at rates exceeding 0.5-0.75 Gm. (approximately 10-15 ml. of a 5% solution)/Kg./hour, although somewhat slower rates are to be preferred.

It follows that the higher the concentration of intravenous dextrose solution, the more slowly it must be given. In many clinical situations where the oral route is not available, additional required calories may be given in this way. Examples are when parenteral nutrition must be maintained over a prolonged period or when maximum protein-sparing action is essential, as in the case of patients with acute renal insufficiency who can be given only very small quantities of fluid (see p. 99). In these instances, hypertonic (25-50%) solutions of dextrose are used. Phlebitis can be minimized by

administering hypertonic solutions very slowly through a small poly-
ethylene catheter advanced through an arm or leg vein as far as
possible into a large vessel.

Fructose has been recommended as a substitute for dextrose in
intravenous nutrition because it disappears from the blood faster
than dextrose and thus can be administered more rapidly and is less
readily lost into the urine. In most situations, however, these ad-
vantages do not seem to be sufficiently impressive to recommend its
general use as a substitute for dextrose. The caloric contributions
of the two sugars are the same.

Ethyl Alcohol.

Ethyl alcohol may be added to dextrose solutions as an addition-
al source of calories. It is metabolized to provide 5.6 Calories/ml.
or 7 Calories/Gm. However, alcohol should not be given intra-
venously in concentrations much exceeding 5% (50 ml. 95% alcohol/
L.), which means that only an additional 280 Calories/L. can be
administered in this way.

Patients receiving alcohol-containing solutions intravenously
must be carefully attended, and the possibility of undesirable reac-
tions with other drugs that are being used must also be considered.

Fat.

From a nutritional standpoint, the most satisfactory concen-
trated source of calories is fat. An emulsion of fat suitable for
intravenous administration is now available as Lipomul I.V.®, a
15% emulsion of cottonseed oil containing also 4.5% dextrose, which
provides 940 Calories in a 600 ml. unit. It is probably not neces-
sary to include fat in the intravenous regimen unless parenteral nu-
trition must be used for a prolonged period. In such circumstances,
adults should receive at least one 600 ml. bottle per day. The fat
emulsion must be given more slowly than dextrose, but it does not
damage the veins. Some patients react with slight elevations of
temperature, which subside if the rate of infusion is slowed   It
occasionally happens that a reaction to the first infusion will occur
but that subsequent infusions from the same lot of emulsion may be
given without incident. Intravenous fat may be contraindicated or
at least must be used with extreme caution in patients whose liver
function is severely depressed.

A so-called "overloading syndrome" has been reported in a
few patients given more than 16 units of the fat emulsion over a
period of 10-14 days. Manifestations included the development of
blood clotting defects and signs of accumulation of lipid in various
organs. All of the findings were reversible when the infusions were
stopped, and no fatalities directly attributable to this syndrome have
been reported. Normally, the serum of patients receiving intra-
venous fat emulsions becomes clear by 12 hours after termination
of an infusion of fat. Lipemia which persists for a longer period is
a sign of overloading and indication for discontinuing the adminis-
tration of fat. In any event it is probably well to limit fat adminis-
tration to 16 units for any patient and to discontinue its use if
lipemia persists 12 hours after an infusion has been completed.

Protein.

When only the parenteral route can be used, the most efficient way to provide nutrients for synthesis of protein is by the administration of amino acid mixtures prepared by enzymatic or acid hydrolysis of casein or fibrin.* The principal disadvantage to the use of intravenous amino acid preparations is that adequate amounts of "protein-sparing" calories from carbohydrates and fat must be administered simultaneously. There is evidence that as much as 53-60 Calories/Kg. may be necessary to attain nitrogen balance when amino acids are used as the sole source of nitrogen, even by mouth, as compared to 35 Calories/Kg. when equivalent amounts of nitrogen are given as whole protein. However, if an adequate caloric intake can be provided (as may now be possible by the use of parenteral fat emulsion), amino acid mixtures are very satisfactory as a source of nitrogen for protein synthesis.

Intravenously administered amino acids are not lost into the urine in any significant quantity.

Most patients tolerate these mixtures well if too rapid administration is avoided. An optimal rate of administration is approximately 300 ml./hour of a 5% solution. It may be assumed that 1 L. of a 5% amino acid solution contributes the equivalent of about 35 Gm. of protein to the diet. Most amino acid preparations contain also 5% dextrose. It is desirable to give the amino acids simultaneously with dextrose.

Sample Regimen: The Three Principal Nutrients.

The following regimen is cited as an example of what is required in parenteral feeding to supply a modest daily intake of protein and calories (in an amount of fluid which is slightly more than the ideal amount). The total infusion time required is about 12 hours.

1000 ml.	5% dextrose and 5% ethyl alcohol	- 480 Calories
2000 ml.	5% dextrose - 5% amino acids	- 680 Calories
600 ml.	15% fat - 4.5% dextrose	- 940 Calories

Totals - 3600 ml. fluid
2100 Calories
70 Gm. protein equivalent

TUBE FEEDING

Feeding by nasogastric tube is occasionally necessary to prevent serious malnutrition, e.g., in severe anoxia, coma, obstructive lesions of the esophagus, fractures of the jaw, and after severe burns.

When liquid formulas are used, very small polyethylene naso-

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*Although plasma or purified protein fractions of the plasma, such as albumin, can be given intravenously, these substances are metabolized slowly and in a manner which is nutritionally uneconomical. Consequently, these substances should not be used for nutritional purposes alone.

gastric tubes (e.g., K-30) which may be left in place for many days are suitable. These tubes may even be used for supplemental feedings in patients who can ingest some of their meals in the usual manner (such as burned patients who may be unable to eat adequate quantities of protein).

In preparing formulas for tube feeding it will probably be necessary to avoid the use of simple sugars, such as dextrose, and too much fat, such as cream, since both tend to cause diarrhea in tube-fed patients. The preferred source of carbohydrate is a dextrinized starch, such as Dexin® or Dextri-Maltose®. A simple formula for feeding by gastric tube is as follows:

Homogenized milk	2200 ml.
Half-and-half (cream and milk)	600 ml
Eggs	6
Dextri-Maltose®	7 Tbsp.

(In a total volume of 3000 ml this mixture contains 120 Gm. protein and 3000 Calories.)

Homogenized milk alone is often the simplest substance to use at the beginning of a tube feeding regimen. It contains 3.5 Gm. of protein and about 70 Calories/100 ml. During the first few days after an operation, the initial rate of feeding should not exceed 50 ml./hour. Thereafter the rate may be increased gradually in accordance with the patient's tolerance until a maximum of 150-200 ml. every 2 hours is reached, usually about 1 week later. If, at this time, the patient is tolerating the feedings well, the homogenized milk may be fortified with 50 Gm. of a powdered protein hydrolysate and 30 Gm. of Dextri-Maltose® or Dexin®. If this is well tolerated, the carbohydrate may be gradually increased over a three-day period to 60 Gm./L. This final formula contributes 160 Gm. of protein and about 2500 Calories in 2400 ml. Vitamins may be added to the formula, using a concentrated solution similar in composition to that given on p. 87.

The formulas and rates of administration described above may also be used for jejunostomy feedings.

A more concentrated tube feeding mixture can be used with the aid of a pump to maintain the flow of the mixture. A sample formula which utilizes commercially available puréed infant foods is as follows:

	Protein (Gm.)	Calories
3 1/2 oz. strained beef	14.6	103
4 1/2 oz. beets	1.5	51
3 raw eggs	18.0	225
Homogenized milk (640 ml.)	23.3	466
Totals	57.4	845

Other meats and vegetables can be used to vary the formula.

Precautions in Tube Feedings.

1. If given rapidly by gravity drip or by injection into the gastric tube, the formula should first be warmed to body temperature.

2. Do not give over 200 ml. at a time.
3. Use special care to prevent aspiration in unconscious patients.
4. Supply sufficient water in addition to the formula to permit excretion of nitrogenous end products. This is particularly important if the formula is high in protein.
5. When stopping or starting a feeding, or whenever the question of gastric retention arises, aspirate the gastric tube and wash it out with water.
6. Avoid constipation and fecal impaction in debilitated patients, if necessary by introducing 30 ml. of milk of magnesia into the tube.

THE NORMAL DIET

Parenteral and tube feedings should be discontinued as soon as possible in favor of a full normal diet taken by mouth. Foods should be palatable and attractively prepared as a stimulus to appetite. The physician should not overlook his responsibilities in this matter since a properly compounded nutritional prescription can be just as important to his patient's welfare as the drugs he may order.

The daily diet for the convalescent adult patient should contain 3000-3500 Calories and 100-150 Gm. of protein. It is not sufficient merely to place these nutrients on a tray within the patient's reach; care must be taken to make certain that the entire nutritional prescription is actually consumed.

It is often difficult for surgical patients to eat large amounts of high-protein foods such as muscle meats. Intermittent feedings may have to be given, but must be carefully spaced so as not to spoil the patient's appetite for regular meals. Fruit juices and broths are of little value as supplementary nutrients because they contribute practically nothing toward remedying protein and caloric deficits, the major dietary faults in need of correction; in fact, these liquid supplements may actually dull the appetite and thus indirectly impair nutrition.

A simple but useful supplementary drink may be prepared by stirring 50 Gm. of skimmed milk powder into 200 ml. of water. This supplies 17 Gm. of protein and 26 Gm. of carbohydrate, more than is available in 1 pint of milk but in only half the volume. Six glasses will supply over 100 Gm. of protein of excellent nutritional quality. Other valuable protein supplements are ice cream, cottage cheese, and eggs.

The "Basic" Foods.

As a guide to the formulation of an adequate diet, the various foods have been arranged into 7 "basic" groups, each of which makes a major contribution to the diet. To secure an adequate diet, some foods from each of the 7 groups should be taken each day as follows:

1. Milk - Two or more glasses daily for adults; 3-4 or more glasses daily for children.
2. Vegetables - Two or more servings daily (in addition to potatoes); 1 vegetable, raw; green and yellow vegetables served often.

3. Fruits - Two or more servings daily; 1 a citrus fruit or tomato.
4. Eggs - Three to 5 a week; 1 daily preferred.
5. Meats, cheese, fish, or legumes - One or more servings daily.
6. Cereal or bread - Most of it whole grain or "enriched."
7. Butter - Two or more tablespoons daily.

SPECIAL DIETS FOR SURGICAL PATIENTS

Surgical diets are listed below according to the purpose for which they are designed. Therapeutic diets are modifications of the Regular or House Diet devised to meet the unusual demands imposed by the illness under treatment. The increased nutritional requirements created by many surgical conditions must be kept in mind. Inadequate diets should be used for only the briefest possible time, and vitamin supplements should be prescribed with them. When the oral intake is insufficient, parenteral supplementation should be considered.

Difficulty with mastication may prevent ingestion of the pre-scribed diet and must be considered in edentulous patients or those with oral neoplasms. A "mechanical" or puréed regular diet will be nutritionally complete.

Types of Surgical Diets

Diet	Indications
Regular or house	Normal maintenance.
High-protein, high-caloric, high-vitamin	Malnutrition; increased nutritional requirements.
Liquids without milk Liquids with milk Surgical soft	Depressed or disturbed gastro-intestinal function, e.g., post-operatively.
Postgastrectomy	After gastric resection or gastro-enterostomy.
Peptic ulcer regimens	Peptic ulcer.
Low-fat	Biliary tract disease.
Minimal residue	Colon preparation, rectal surgery, colitis.
Low-sodium	Cardiac disease, e.g., congestive failure; edema.
Gastric tube feeding	Comatose, uncooperative, mal-nourished, or severely anorexic patients; markedly increased nutritional demands, e.g., burns; gastrostomy.
Jejunostomy feeding	Jejunostomy.

High-protein, High-calorie, High-vitamin Diet.

This diet is prescribed for malnourished patients or those with increased requirements, as in hyperthyroidism, febrile illness, and surgical convalescence. At least 3000-3500 Calories and 100-150 Gm. of protein should be provided.

Composition: All the basic foods with increased amounts of meat, liver, fish, poultry, eggs, milk, cheese, whole grain cereals, carrots, green vegetables, citrus fruits, and butter or margarine. Recipes and drinks may be fortified with such protein sources as skimmed milk powder.

Liquid Diet Without Milk.

Liquid and soft diets are suitable after injury or surgery when the function of the gastrointestinal tract is impaired. Under these circumstances the transition to a regular diet must be made in gradual stages when anorexia, nausea, vomiting, and paralytic ileus subside. The absence of distention, the return of peristalsis, and the passage of flatus are indications that the diet may be advanced.

Composition: Cereal gruel made with water, clear broth, bouillon, tea, coffee, plain jello, sugar, and strained fruit juice.

Liquid Diet With Milk.

Composition: Broth, bouillon, cereal gruel made with milk, strained creamed soups lightly seasoned, tomato juice, strained fruit juice, lemonade, sherbet, ice cream, plain jello, junket, all milk and cream beverages, cocoa, ginger-ale, carbonated beverages, coffee, tea.

Surgical Soft Diet.

This diet is transitional between the liquid and regular diets. The basic foods are included in each day's menu, but only those are selected which are lowest in cellulose and connective tissue. All foods are bland, smooth, and easily digested; stimulating factors (acids, extractives, concentrated carbohydrates, and condiments) are kept to a minimum. When served in 3 meals with 3 between-meal feedings, the surgical soft diet qualifies as a "six-meal bland diet" suitable for patients with peptic ulcer, gastritis, hiatus hernia, and after gastric resection.

Composition: Lean meat, fish, poultry, eggs, milk, mild cheese, potatoes; cooked, tender, or puréed vegetables and fruits; refined cereals and breads, custards, puddings, gelatin desserts, ice cream, plain cake, cream, butter, margarine, salt, and sugar in moderation.

Restrictions: Fried foods, meat soups and gravies, raw or coarse vegetables and fruits, whole grain cereals or bread, spices, highly seasoned foods, rich desserts and pastries, coffee, tea, carbonated and alcoholic beverages.

Postgastrectomy Diet.

It is likely that postgastrectomy patients will be maintained exclusively by parenteral feeding for at least the first 3 postoperative days or until bowel sounds return. When oral feeding can be instituted, it is important to gradually increase the quantity of small, frequent, bland, low-residue feedings. Undue distention of the stomach must be avoided. Feedings should be reduced or omitted

if nausea, vomiting, or a marked sense of gastric fullness occurs. It may occasionally be possible to advance the diet more rapidly than the following schedule indicates.

Day	Foods Allowed
Day of operation	Nothing by mouth.
First 3 postoperative days	Exclusively on I.V. fluids.
Fourth postoperative day	30 ml. cereal gruel or water every hour.
Fifth postoperative day	60 ml. cereal gruel or water every hour.
Sixth postoperative day	90 ml. cereal gruel, milk, or water every hour.
Seventh postoperative day	120 ml. strained cereal with sugar and cream, malted milk, or baked potato mashed with hot milk every 2 hours. Water ad lib.
Eighth postoperative day	Same as seventh day plus 2 crackers.
Ninth postoperative day	180 ml. strained cereal with sugar and cream, creamed soup, eggnog, coddled eggs, custard, soft cooked cereals every 3 hours.
Tenth postoperative day	Same as ninth day or proceed to surgical soft diet served in 6 feedings.

Peptic Ulcer Regimens (Sippy Diets).

Milk, a bland food without cellulose, forms the basis for these diets, which are nonirritating and exert an antacid (buffering) action when taken on a regular schedule. After Stage IV, the six-meal bland diet is appropriate (surgical soft diet; see p. 95).

Composition:

Stage I - 3 oz. (90 ml.) equal parts milk and cream every hour from 7:00 a.m. to 7:00 p.m. Night feedings if necessary.

Stage II - Stage I plus addition of fine-grain cereals, cooked eggs, toast, butter, mashed potatoes. Give these foods as 1 additional serving the first day, 2 the second day, etc., and as desired by the fourth day. Orange and fruit juices may be taken as desired if distaste for milk occurs.

Stage III - Three meals daily with 6 oz. (120 ml.) milk and cream mixture at 10:00 a.m., 2:00 p.m., and 8:00 p.m.; add to Stage II puréed vegetables, cottage cheese, rice, pastes, plain puddings, gelatin desserts. Alkalies are given on alternate hours between meals and formula. AVOID meats, fish, poultry, cheese (other than cottage cheese), raw vegetables, fried foods, spices and condiments, meat soups, tea, or coffee.

Stage IV - Stage II plus 3-4 servings weekly of boiled or broiled beef, lamb, chicken, or fish.

Restrictions: As for surgical soft diet (see p. 95).

Low-fat, Non-gas-forming Diet.

The basic foods are included in each day's menu, but those of low fat content are selected. Fat intake should be reduced to 30-50 Gm./day. The energy requirements are made up by increasing the protein and carbohydrate intake. Because of the reduced fat intake and the associated restrictions in fat-soluble vitamins, fruits and vegetables high in vitamin A should be served daily.

Composition: Lean meat, fish, poultry, skimmed milk or buttermilk, cottage cheese, cereal products, bread, vegetables, fruits (except those listed below), gelatin desserts, sherbet, puddings without cream, sugars, jellies.

Restrictions: Pork, ham, bacon, fatty cuts of meat, cream, cabbage family, onions, turnips, cucumbers, radishes, green peppers, dried beans and peas, melons, raw apples, butter, margarine, mayonnaise, oil, nuts, chocolate, fried foods, pastries, and highly seasoned foods.

Minimal Residue Diet.

This diet is planned to leave a small residue after digestion. It is useful during preoperative and postoperative periods when it is desirable to reduce the fecal residue to prevent bowel movements for several days (as after rectal surgery). This diet is inadequate and should be used as briefly as possible. The surgical soft diet is also low in residue and can be made even more so by the omission of milk. It should be prescribed when a less restrictive regimen is satisfactory.

Composition: Eggs (soft-cooked, poached, or hard-cooked), meats (tender beef, veal, liver, fowl, oysters, sweetbreads), cereals (farina made with water, rice, rice-products), soda crackers, zwiebach, arrowroot wafers, desserts (jello, arrowroot cookies, gelatin flavored with coffee or carbonated beverages), clear broths, bouillon, carbonated beverages, tea, coffee, butter, sugar, pure sugar candies, and jelly.

Restrictions: All foods not listed.

Low-sodium Diet.

The low-sodium diet is used in those conditions associated with generalized edema, with or without ascites, secondary to chronic heart failure or hepatic insufficiency. In the preparation of such patients for surgical intervention and during the postoperative period, sodium restriction may be indicated. The best therapeutic results are obtained with diets containing less than 0.5 Gm. of sodium.

Each of the following low-sodium diets contains 2000 Calories They are the same in composition except for the beverage.

250 mg. sodium diet: use Lonalac® as beverage.
500 mg. sodium diet: use whole milk as beverage.

Breakfast:

Fruit	$1/2$ cup
Salt-free cooked or "puffed" cereal	$1/2$ cup
Salt-free bread	1 slice
Salt-free butter or margarine	2 tsp. (1 pat)
Egg	1
Lonalac® or whole milk	$1/2$ cup

Noon and Evening Meals:

Salt-free fresh meat	$3^{1}/2$ oz.
Salt-free potato or rice	$1/2$ cup
Salt-free cooked or raw vegetables	As desired
Salt-free bread	1 slice
Salt-free butter or margarine	2 tsp. (1 pat)
Fruit	$1/2$ cup
Lonalac® or whole milk	8 oz.

Restrictions:

1. Ham, bacon, bacon fat, salt pork, corned beef or pork, luncheon meats, canned meats, fish or poultry.
2. Prepared cereals with salt, "quick-cooking" cereals, breads leavened with baking powder or baking soda.
3. Prepared foods or prepared desserts.
4. Canned vegetables, dried fruits, commercial salad dressings, catsup.
5. Salted nuts, salted popcorn, potato chips.
6. Salt, garlic salt, onion salt, celery salt, baking powder, baking soda.
7. Celery, olives, pickles, relishes, chard.
8. To avoid distention - Cabbage family, onions, turnips, peppers, dried beans, cucumbers, sweet potatoes, raw apples, melons.

Instructions:

1. Lonalac® is a low-sodium milk from which the sodium has been removed by dialysis. One-half cup of the dried powder is mixed with 2 cups of water, and, if desired, flavored with chocolate.
2. To make salt-free margarine or butter, wash and knead in 5 changes of cold water.
3. Use fresh or frozen vegetables or special "salt-free" canned vegetables. May use artichokes, beets, carrots, spinach, and other greens twice weekly.
4. Use only fresh or cooked fruit.
5. Use only granulated gelatin in salads and desserts.
6. May use pepper, herbs, and other spices.
7. May use 1 of the sodium-free salt substitutes.

5 . . .

Fluid and Electrolyte Therapy

WATER METABOLISM

BODY WATER AND ITS DISTRIBUTION

The total body water comprises 40-65% of total body weight (avg., 55% for adult men and 47% for adult women). The body water is distributed throughout 2 main compartments: the extracellular compartment (plasma and interstitial fluid), which contains about one-fourth of the total body water; and the intracellular compartment, which contains about three-fourths of the total body water.

Distribution of Body Water in the Male

Fluid Compartment	Per Cent of Body Weight	ml. of Water in a 154 lb. (70 Kg.) Man
Extracellular water		
Plasma	4-5 %	3,200
Interstitial fluid	11-12%	7,300
Intracellular water	40-45%	31,500

The considerable variation in percentage of body weight which is water is due mainly to the effect of body composition on the water content of the tissues. The higher the fat content of a given subject, the smaller the percentage of his body weight which is water. If a correction for the fat content of the body is made, the total body water in various subjects is relatively constant when expressed as a percentage of the so-called lean body mass, i.e., the sum of the fat-free tissue. Consideration should be given to this fact when calculating fluid requirements on the basis of body weight in order to avoid excess administration of water to obese patients.

NORMAL WATER LOSSES AND WATER REQUIREMENTS

Water is lost from the body by 4 routes: from the skin, as sensible and insensible perspiration; from the lungs, as water vapor in the expired air; from the kidneys, as urine; and from the intestines, with the feces. In the absence of visible perspiration, the total losses from the skin and the lungs are generally referred to as the "insensible loss." In adults, this loss is considered to be 0.5 ml./Kg./hour (12 ml./Kg./day), i.e., approximately 800 ml./day for the average adult. In children, the insensible loss is somewhat higher when estimated on the basis of body weight, varying from 1.3 ml./Kg./hour in infants to 0.6 ml./Kg./hour in the older child.

The normal daily water losses and water allowances are sum-
marized in the following table:

Daily Water Losses and Water Requirements
for Normal Individuals Who Are Not Working or Sweating

	Losses				Requirements	
	Urine (ml.)	Stool (ml.)	Insensi-ble (ml.)	Total (ml.)	ml./ person	ml./ Kg.
Infant (2-10 Kg.)	200-500	25-40	75-300 (1.3 ml./ Kg./hr.)	300-840	330-1000	165-100
Child (10-40 Kg.)	500-800	40-100	300-600	840-1500	1000-1800	100-45
Adolescent or Adult (60 Kg.)	800-1000	100	600-1000 (0.5 ml./ Kg./hr.)	1500-2100	1800-2500	45-30

ADDITIONAL WATER REQUIREMENTS IN DISEASE

Insensible losses may rise much higher than normal postoper-
atively or in febrile or debilitated states. The quantity of fluid lost
from the surface of the body may also be very large in the exten-
sively burned patient.

In high environmental temperatures or when visible sweating
occurs for any reason, additional water must also be provided to
replace the additional losses of sensible perspiration. It is diffi-
cult to estimate these losses accurately. During moderate sweating
the fluid lost is about 300-500 ml./day, but in cases where extreme
sweating occurs the losses may exceed 2000-3000 ml./day.

Water loss from the gastrointestinal tract is negligible under
normal circumstances. However, this route of loss may assume
great importance in patients with prolonged diarrhea or vomiting,
in cases where nasogastric suction is in use, or where there is
drainage from fistulas or an ileostomy.

In kidney disease in which the ability to form a concentrated
urine may be impaired, such as chronic nephritis, nephrosclerosis,
or pyelonephritis, it may be necessary to provide additional fluid.
This is illustrated in the table below, which relates the daily volume
of urine necessary for removal of metabolic wastes to the concen-
trating power of the kidney expressed in terms of the sp. gr. of the
urine. On a regular diet, 50-60 Gm. of metabolic wastes must be
excreted daily (approximately 1.5-2 Gm./100 Calories metabolized).
This may be reduced to 30-35 Gm./day by decreasing the protein
intake. From the table it is evident that a patient on a normal diet
who is unable to form a urine more concentrated than sp. gr. 1.010-
1.014 must be given enough fluid to permit a daily urine output of as
much as 2100 ml. if uremia is to be avoided.

**Relation Between Urine Volume Necessary to Remove
Metabolic Wastes and Concentrating Power of Kidney
(Expressed as Sp. Gr. of the Urine)**

Sp. Gr. of Urine	Urine Volume	
	35 Gm. Load of Metabolic Wastes	50 Gm. Load of Metabolic Wastes
1.032-1.029	500	700
1.028-1.025	600	850
1.024-1.020	700	1000
1.019-1.015	850	1200
1.014-1.010	1200	2100

ESTIMATION OF WATER LOSSES

An accurate record of the quantity and source of the fluid lost
by all routes is of utmost importance for proper replacement ther-
apy. This ordinarily includes measurement of daily urinary ex-
cretion and of any fluid recovered from the gastrointestinal tract.
It is often difficult to obtain an accurate measurement of total fluid
losses when a major category of loss is from perspiration, from
exudation (as in burns), from diarrhea, or when fluid is lost into
dressings. In such circumstances, daily weighing of the patient is
of great value. In many hospitals special bedside scales are avail-
able for patients who cannot be weighed on the usual scales.

Patients maintained on parenteral fluids (and seriously ill
patients generally) cannot be expected to gain weight unless they are
overhydrated. Properly hydrated adult patients should normally
lose 0.5-1 lb./day until adequate nutrition can be established during
convalescence. If rapid losses in weight occur, the patient must be
presumed to be dehydrated. The table below shows the approximate
relationship between the extent of a rapidly occurring weight loss
and the degree of dehydration.

Relation of Acute Weight Loss to Degree of Dehydration

Weight Loss Expressed as % of Normal (or Preoperative) Body Weight	Degree of Dehydration
Loss of 4% body weight	Mild
Loss of 6% body weight	Moderate
Loss of 8% body weight	Severe

Because such rapidly occurring weight losses are due mainly to
loss of fluid, the extent of the loss may be used to estimate the
amount of fluid to be replaced. Each Kg. of weight loss is assumed
to be equivalent to 1 L. of fluid.

An exception to the foregoing remarks concerning the relation-
ship between acute weight loss and dehydration must be made in the
case of receding edema or a diuresis after administration of diuretic
agents.

FLUID REQUIREMENTS IN THE IMMEDIATE
POSTOPERATIVE PERIOD

During the immediate postoperative period the posterior pituitary and the adrenal cortex, in responding to the stress of the operation, cause excessive retention of water (increased antidiuretic effect) and sodium. Urinary output then becomes a poor guide to the need for fluid; in fact, oliguria at this time may be considered a normal physiologic response, particularly after severe stress and/or trauma. Additional fluid given under these circumstances in an effort to increase the urinary output will not only be ineffective but may actually result in serious overhydration. It is therefore recommended that during the first postoperative day conservative amounts of water (e. g., up to 1500 ml./day for an adult) and no electrolyte be given unless significant gastrointestinal losses have occurred, in which case small amounts (50-75 mEq.) of sodium salts may be given. As noted above, patients can be expected to lose 0.5-1 lb./day during the first few days after surgery. The absence of a weight loss or a gain in weight is strong presumptive evidence for overhydration.

INTERNAL LOSSES OF FLUID AND ELECTROLYTE

The extracellular fluid space may be depleted of both fluid and electrolyte by losses into fluid spaces newly created by a disease process. Examples are the accumulation of fluid in the edema of burns; in an area of infection, as within a serous cavity; or in the intestine during ileus. The fluid and electrolyte thus lost results in what may be termed "internal dehydration." Unless prompt and adequate replacement of these losses is made, the effect on the circulating plasma volume will be just as serious as if the losses had been external.

These fluids will be reabsorbed later unless they have been removed (as by suction in a case of ileus), and the replacement of fluid and electrolyte must be reduced to compensate for this. In the case of a burn, resolution of fluid pooled outside the circulation usually begins after 48-72 hours, whereas a longer period is required in the case of infection or trauma.

ELECTROLYTE METABOLISM

ELECTROLYTE COMPOSITION OF BODY FLUIDS

The table below lists the concentrations of the various inorganic salts (electrolytes) in the plasma and in the cells. It will be noted that the electrolyte composition of intracellular fluid differs from that of the plasma in that potassium (rather than sodium) is the principal cation, and phosphate (rather than chloride) is the principal anion. The amount of protein within the cell is also considerably larger than that in its extracellular environment.

Normal Electrolyte Composition of Body Fluids

	Atomic or Radicular Weight	mEq. Wt. (mg.)	Extracellular Fluid (Plasma)			Intracellular Fluid (Muscle) mEq./L. (Avg.)
			mEq./L.		mg./100 ml.	
			(Avg.)	(Range)		
Cations						
Na$^+$	23	23	143	135-147	310-340	13
K$^+$	39	39	5	4.6-5.6	18-22	140
Ca^{++}	40	20	5	4.5-5.5	9-11	Trace
Mg^{++}	24	12	2	1.5-3.0	1.8-3.6	45
Total			155			198
Anions						
Cl$^-$	35	35	103	100-112	350-390	3
(As NaCl)	(58)	(58)			(590-660)	
HCO$_3^-$*	-	-	27	25-30	56-65†	10
HPO$_4^-$ (as P)‡	31	17.2	2	1.8-2.3	3-4	100
SO$_4$ (as S)	32	16	1			20
Org. Acids§	-	-	6			Trace
Protein§	-	-	16			65
Total			155			198

*HCO$_3^-$ is measured as CO$_2$ content and frequently reported in Vol.% (ml./100 ml. plasma). To convert Vol.% of CO$_2$ to mEq./L. HCO$_3^-$, divide Vol.% by 2.24.
†Vol.%.
‡The inorganic phosphorus in the serum exists as a buffer mixture in which approximately 80% is in the form of HPO$_4^-$ and 20% as H$_2$PO$_4^-$. For this reason the mEq. weight is usually calculated by dividing the atomic weight of phosphorus by 1.8. Thus, the mEq. weight for phosphorus in the serum is taken as 31/1.8 = 17.2.
§The organic acids and the proteins are expressed in terms of their combining power with cations. For protein, the cation equivalence in mEq. is calculated by multiplying the number of grams of total protein/100 ml. by 2.43.

    The chemical reactivity of the various electrolytes in the body fluids cannot be evaluated when their concentrations are given only in accordance with their weights in a given volume (e. g., mg./100 ml.) any more than the work performance (horsepower) of an electric motor can be expressed merely in terms of its weight. In order to convert electrolyte concentrations to a common unit which expresses the individual contribution of each to the total chemical reactivity of all, it is necessary to express their concentrations in mEq./unit of volume.

    The mEq. weight of an element is simply its atomic weight divided by its valence (number of electric changes carried by the element). In the above table the mEq. weights of the electrolytes of the body fluids are listed. To convert a concentration of an electrolyte from mg./100 ml. to mEq./L., the formula shown below is used:

$$\frac{mg./100 \ ml. \times 10}{mEq. \ weight} \quad mEq./L.$$

For example, if the concentration of plasma sodium is reported as 322 mg./100 ml., the equivalent concentration is 140 mEq./L.:

$$\frac{322 \times 10}{23} = 140 \text{ mEq./L.}$$

COMPOSITION OF GASTROINTESTINAL SECRETIONS AND SWEAT

The volume and composition of gastrointestinal secretions and sweat are shown in the table below. It will be noted that large quantities of fluid and electrolyte are normally secreted into the gastrointestinal tract but that almost all are subsequently reabsorbed (mainly in the colon), as evidenced by the normal minimal losses of fluid and electrolyte in the feces. When reabsorption is impaired, as may occur during vomiting, diarrhea, obstruction, or a fistulous process such as a biliary or pancreatic fistula, or when fluid is removed by nasogastric suction or is lost in a recent ileostomy, depletion of fluid and electrolyte can occur rapidly. **Note:** In surgical patients such gastrointestinal losses are the principal causes of severe dehydration and electrolyte depletion.

Other disorders in which disturbances of fluid and electrolyte metabolism are prominent include diabetes mellitus, congestive heart failure, hepatic cirrhosis, Addison's disease, kidney disease, extensive burns, and shock.

Volume and Composition
of Gastrointestinal Secretions and Sweat*

Fluid	Avg. Volume (ml. /24 hr.)	Electrolyte Concentrations (mEq./L.)			
		Na^+	K^+	Cl^-	HCO_3^-
Blood plasma†		135-150	3.6-5.5	100-105	24.6-28.8
Gastric juice	2500	31-90	4.3-12	52-124	0
Bile	700-1000	134-156	3.9-6.3	83-110	38
Pancreatic juice	>1000	113-153	2.6-7.4	54-95	110
Small bowel (Miller-Abbott suction)	3000	72-120	3.5-6.8	69-127	30
Ileostomy Recent	100-4000	112-142	4.5-14	93-122	30
Adapted	100-500	50	3	20	15-30
Cecostomy	100-3000	48-116	11.1-28.3	35-70	15
Feces	100	<10	<10	<15	<15
Sweat	500-4000	30-70	0-5	30-70	0

ACID-BASE BALANCE

The laboratory detection of disturbances of acid-base balance is best accomplished by measurement of the pH of the arterial blood and of the total CO_2 in the plasma (plasma CO_2 content). Carbonic acid (H_2CO_3) and bicarbonate (HCO_3^-)(as well as small amounts of physically dissolved CO_2, which need not be considered) constitute the measured total CO_2 of the plasma. When the pH of the blood is normal (pH 7.4), it may be assumed that 1 part of H_2CO_3 is present for each 20 parts of HCO_3^-. As long as this ratio is maintained, the pH of the blood remains normal.

RESPIRATORY ACIDOSIS AND ALKALOSIS

The content of H_2CO_3 in the blood is under the control of the respiratory system because of the effect of respiratory elimination of CO_2 on the following reaction·

$$CO_2 + H_2O \rightleftharpoons H_2CO_3$$
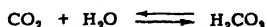
Disturbances in acid-base balance which are due to alterations in content of H_2CO_3 in the blood are said to be respiratory in origin. Thus, respiratory acidosis will occur if CO_2 and consequently H_2CO_3 accumulate in the blood. Examples are pneumonia, emphysema, congestive failure, asthma, and inadequate ventilation during anesthesia and in the postanesthetic period. Depression of the respiratory center, as by overdosage of morphine or other respiratory depressants, may also be a cause of respiratory acidosis.

Compensation for respiratory acidosis occurs by an increase in plasma bicarbonate to restore the normal 1:20 ratio between carbonic acid and bicarbonate at which the blood pH is normal. The total CO_2 is therefore elevated in compensated respiratory acidosis.

Respiratory alkalosis will occur when the rate of elimination of CO_2 is excessive so that blood H_2CO_3 is reduced. This may be brought about by voluntary or forced hyperventilation. Examples are hysterical hyperventilation, CNS disease affecting the respiratory system, the early stages of salicylate poisoning, the hyperpnea occurring at high altitudes, or injudicious use of respirators. Respiratory alkalosis may also occur in patients in hepatic coma.

In respiratory alkalosis compensation occurs by a decrease in plasma bicarbonate; the total CO_2 is therefore decreased in compensated respiratory alkalosis.

These changes are illustrated in the chart on p. 106.

METABOLIC ACIDOSIS AND ALKALOSIS

Disturbances in acid-base balance which are due to alterations in content of bicarbonate in the blood are said to be metabolic in origin. A deficit of bicarbonate will produce a metabolic acidosis; an excess of bicarbonate, a metabolic alkalosis. Compensation occurs by adjustments of the H_2CO_3 concentration - in the first

corrected. Conversely, retention of sodium or its administration in excess is usually associated with overhydration, often detectable by the appearance of excess extracellular fluid (i.e., edema).

The effect of excess sodium in producing and maintaining edema is enhanced in the presence of hypoproteinemic states (particularly hypoalbuminemia). Hypoproteinemia commonly occurs in chronic disease (e.g., cirrhosis), in malnourished patients (who may come to surgery in a semi-starved condition), and in patients for whom adequate nutrition must be unduly postponed during the postoperative period.

Mobilization of excess body sodium and restriction of sodium intake is essential to reduce the overhydration of edematous patients. This may be accomplished by the use of diuretic agents, which increase elimination of sodium salts in the urine; in severely hypoproteinemic patients, it may also be useful to administer salt-poor albumin to aid in mobilizing edema fluid.

INTERPRETATION OF THE SERUM SODIUM

Measurement of the serum sodium is frequently employed in an effort to ascertain the status of a patient with regard to sodium and thus to investigate an important phase of water balance as well as acid-base balance. However, such a measurement does not necessarily reflect total body sodium because the amount of water in the body affects the concentration of many substances in the plasma, including the plasma electrolytes. Serum sodium determinations should thus be interpreted (in the light of the clinical circumstances) as a reflection of the relationship between sodium and water. The insight thus gained into the sodium status of the patient can be used in determining the need for (1) administering or withholding sodium, in cases where the serum sodium is low or normal, (2) administering more sodium than water (hypertonic solutions), or (3) rigidly restricting sodium and rapidly administering sodium-free fluids, as in hypernatremic states.

The relationship of serum sodium concentration to total body sodium in various clinical states is summarized on p. 110. It will be noted that a serum sodium may be low in the presence of high, normal, or low total body sodium. Similar variations are possible with a normal or high serum sodium. It is therefore important to correlate measurements of serum sodium with pertinent clinical observations in order to utilize laboratory data as a proper guide to fluid and electrolyte therapy.

As already noted, edema is a sign of an increase in body water. In edematous states, regardless of the apparent concentration of sodium in the serum, it can be concluded that the total body sodium is actually increased. Electrolyte therapy to raise the serum sodium is not indicated.

Low serum sodium in the presence of a normal total body sodium, which is a sign of excess water intake, is a feature of so-called "dilutional hyponatremia." If excessive amounts of water are given rapidly, water intoxication will occur.[*] These syndromes are observed in surgical patients given excessive amounts

[*]A useful diagnostic aid in the dilutional states is the measurement of Hgb. and PCV on the same sample of blood. A normal Hgb. appears to be low, whereas the PCV will appear to be high. The Hgb.

of electrolyte-free fluids, particularly in the immediate postoperative period when water requirements are reduced because of the retention of fluid which normally occurs as a result of stress (see p. 102).

In mild dilutional hyponatremia, restriction of fluid may be all that is required to return the serum sodium to normal. If signs of acute water intoxication are apparent, hypertonic (3-6%) solutions of sodium chloride may be needed.

The therapy of hyponatremic states with low total body sodium usually requires both salt and water because the patient is both salt-depleted and dehydrated. Isotonic saline solutions are therefore used for replacement therapy of these patients.

The patient with a high serum sodium is usually water depleted, so that water by mouth (or dextrose and water by vein) is indicated to return the serum sodium to normal. Electrolyte must be withheld, particularly in cases where the cause of the hypernatremia is excess administration of salt.

METABOLISM OF POTASSIUM

Potassium is the chief cation of the intracellular fluid. Like sodium in the extracellular fluid, it influences acid-base balance and the retention of water. Because potassium is almost entirely an intracellular component, the levels of potassium in the serum may not accurately reflect the true potassium status of the patient. As with sodium, the significance of serum potassium levels must be interpreted by correlation with clinical observations.

High levels of potassium in the serum (hyperkalemia) are not indicative of an excess of total body potassium but rather of excessive loss from the cell accompanied by some degree of failure of the kidney to excrete potassium, as in renal failure, advanced dehydration, or shock. Hyperkalemia may also occur if potassium is administered at an excessive rate, usually by the intravenous route.

The clinical manifestations of hyperkalemia are chiefly cardiac and CNS depression. Cardiac signs include bradycardia and poor heart sounds, followed by peripheral vascular collapse and, ultimately, cardiac arrest. Ecg. changes are characteristic, and include elevated T waves, widening of the QRS complex, progressive lengthening of the P-R interval, and, finally, disappearance of the P wave. Other symptoms commonly associated with elevated extracellular potassium include mental confusion; weakness, numbness, tingling, and flaccid paralysis of the extremities; and weakness of the respiratory muscles.

A low serum potassium (hypokalemia) is almost inevitably an indication of true potassium depletion, but it must be emphasized that an intracellular deficit of potassium may coexist with normal serum levels.

Potassium deficiencies develop readily during the postoperative period when solutions which do not contain potassium are given intravenously for more than a few days. This occurs because these

reflects dilution of the blood; the PCV the swelling of the red cells because of excess accumulation of intracellular water.

RELATIONSHIP OF SERUM SODIUM TO TOTAL BODY SODIUM IN VARIOUS CLINICAL STATES

Serum Sodium	Total Body Sodium	Clinical States	Fluid and Electrolyte Therapy
Low (hyponatremia) < 130 mEq./L.	High	Edematous states (e.g., nephrosis, cirrhosis, cardiac disease). May also occur after severe burns and in the immediate postoperative period.	Not indicated to raise serum sodium.
	Normal	Patients on low sodium intake retaining water as a metabolic response to trauma or surgery, particularly if given excess water (dilution syndrome; water intoxication). May also occur in cirrhotic patients after paracentesis.	Mild: Restrict fluids. Severe: Hypertonic (3-6%) sodium chloride solution may be needed.
	Low	Addison's disease; salt-wasting nephritis; gastrointestinal fluid and electrolyte losses; prolonged sweating with free access to water; perhaps in prolonged use of diuretic agents and on salt-free diets.	Isotonic sodium chloride solution.
Normal 135-145 mEq./L.	High	Renal, cardiac, or hepatic disease; also carcinoma involving pleural or peritoneal cavities. Caused by renal retention of water and salt in the same osmotic ratio.	
	Low	In the early stages of rapid salt depletion from gastrointestinal losses, renal excretion of a dilute urine preserves osmolarity of body fluids. A similar situation prevails in diabetic acidosis.	
High (hypernatremia) > 150 mEq./L.	High	Excess administration of sodium salts.	Water by mouth or dextrose and water I.V. Withhold electrolytes.
	Normal	Simple dehydration due to deprivation of water; diabetes insipidus (congenital, or acquired, as in the diuretic phase of acute renal insufficiency or after cerebral trauma).	
	Low	Prolonged sweating without access to water.	Hypotonic sodium chloride solution.

patients are in negative nitrogen and caloric balance, so that in-
creased quantities of potassium are lost from the cells and then ex-
creted into the urine. Furthermore, all of the gastrointestinal
fluids contain considerable quantities of potassium; removal of
these fluids in nasogastric suction, losses from a fistulous process,
or diarrhea - all enhance potassium depletion.

Alkalosis favors potassium depletion principally by increasing
its excretion into the urine. The adrenocortical hormones, particu-
larly aldosterone, also increase excretion of potassium. Thus the
adrenocortical response to severe stress (as after surgery, trauma,
or severe burns) further contributes to potassium loss.

Certain of the diuretic drugs, such as acetazolamide (Diamox®)
or chlorothiazide (Diuril®), increase potassium excretion. Patients
who come to surgery after treatment with adrenocortical steroids
or diuretic drugs or who have been malnourished and in negative
nitrogen balance for some time must be considered to have a chronic
potassium depletion. Such patients may develop severe signs of
potassium deficiency very early in the postoperative period.

The persistence of alkalosis after adequate replacement of
sodium chloride and water is a strong indication of an underlying
potassium deficiency. Other symptoms of a potassium deficit are
muscle weakness, irritability, and paralysis. Tachycardia and
dilatation of the heart with gallop rhythm are also noted. Character-
istic Ecg. changes in hypokalemia are, first, a flattened T wave;
later, inverted T waves with a sagging ST segment and atrioven-
tricular block, and finally cardiac arrest.

Because of the occasional unreliability of the serum potassium
levels, the Ecg. is of great diagnostic value in that it assesses the
cellular status of this element.

Correlation of the Serum Potassium Concentration and the Electrocardiogram
(Providing there is no parallel change in Na and Ca)*
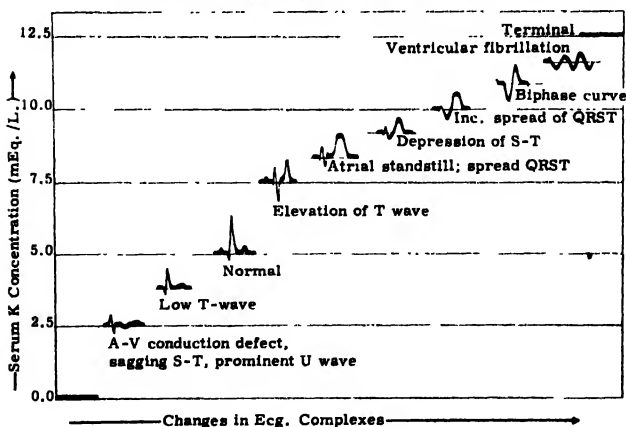
BLOOD AND PLASMA VOLUME

A surgical patient may readily develop a reduction in blood volume because of dehydration, losses of blood and plasma, or chronic undernutrition. Patients who come to surgery with a decreased blood volume are more susceptible to the hypotension which may be induced by anesthetic drugs, or to vascular collapse and shock after even a minimal operative loss of blood. It is therefore important to anticipate this possibility in such patients by a preoperative determination of the blood volume and to correct blood volume deficits before surgery. Because of the wide range of "normal" values for plasma volume (30-65 ml./Kg.) which have been reported, it may also be of value to obtain a measurement of plasma and blood volume preoperatively as a baseline reference for each patient. Such information will be particularly useful if extraordinary losses of blood or plasma require frequent transfusions in the postoperative period. Blood and plasma volume determinations are more useful than measuring simply the PCV or Hct. as a means of assessing the adequacy of replacement of blood or plasma.

MEASUREMENT OF PLASMA AND BLOOD VOLUME

The simplest method of measuring plasma volume is by the use of the blue dye T-1824 (Evans blue). However, the entire blood volume must then be calculated from the Hct. This may not be entirely reliable because the Hct. of the peripheral blood does not necessarily reflect that of the entire blood supply, particularly in pathologic states. For this reason isotopic technics which depend on direct labeling of the red cells are preferred if available. Plasma volume may also be measured by the use of isotopically labeled (I^{131}) human serum albumin. This technic seems to have no particular advantage over the T-1824 method except when repeated determinations at frequent intervals are required.

USE OF BLOOD TRANSFUSIONS

Surgical patients are often found to be anemic, frequently as a result of chronic blood loss. This anemia must be corrected as soon as practicable. In patients who are protein-deficient as well as anemic, the regeneration of Hgb. takes precedence over that of other proteins; consequently, it cannot be expected that the serum proteins will rise until the Hgb. concentration of the blood has first been brought to normal.

Blood transfusions are required to correct anemia only when the loss of blood is acute and/or further bleeding at a rapid rate may be expected. In anemia due to chronic blood loss, iron deficiency is the predominant defect that must be corrected.

Iron deficiency anemia in patients who have sustained chronic losses of blood is of the hypochromic microcytic type. RBC, Hgb., and Hct. determinations are usually sufficient to make a diagnosis of iron deficiency anemia in a patient with a history of chronic blood loss. In these circumstances adequate treatment with iron is all

that is required to correct the anemia. Blood transfusions are not required unless the rate of bleeding is very rapid, immediate surgery is required, or the anemia is in itself more dangerous to the patient than the dangers of transfusion.

Normal persons can probably replace blood at a rate of at least 1000 ml./week when sufficient iron is made available, thus in most patients a blood loss of 500-1000 ml./week can be sustained, and some increase in Hgb. can still occur if adequate amounts of iron are supplied. Iron therapy may have to be continued for 3-5 weeks before the Hgb. levels return to normal. Iron must be given by a parenteral route if the patient cannot tolerate iron salts given orally or if absorption of iron from the intestine is inadequate, as is the case after resection of the stomach or in widespread disease of the small intestine.

In treatment of an iron deficiency anemia, Ferrous Sulfate, U.S.P., may be given in a dose of 200 mg. (3 gr.) orally t.i.d. If oral therapy is not possible, a form of iron which can be administered parenterally should be used. The total amount of iron to be given parenterally is determined by providing 1 Gm. to replenish iron stores plus that represented by the Hgb. deficit. If Hgb. is normal (100%), the total body Hgb. contains 3 Gm. of iron. An iron-deficient patient with a Hgb. of 50% would therefore require 1.5 Gm. to provide for the synthesis of Hgb. to a normal level and 1 Gm. to replenish iron stores (total of 2.5 Gm.). Because of the danger of overdosage when iron is given parenterally, it is important to give only that amount of iron which is required to correct the existing iron deficiency.

POSTOPERATIVE MAINTENANCE

POSTOPERATIVE BLOOD AND PLASMA

Prevention of shock due to excessive blood or plasma loss is of first priority. A 30% reduction in the normal circulating blood volume threatens shock, and even a more moderate blood loss may lead to shock if it occurs rapidly or if it occurs in a patient whose blood volume is already low.

Properly cross-matched whole blood is the preferred replacement fluid in most instances of threatened or actual shock or in the presence of excessive hemorrhage. When whole blood is not available, blood plasma or a plasma expander, such as dextran, may be used instead. In the treatment of shock characterized by a high PCV (as in the early phase of severe burns), these latter substances may be preferred until the PCV begins to fall.

POSTOPERATIVE WATER

An accurate record of the quantity and routes of fluid loss is essential to proper replacement therapy. On the basis of such a record, fluids (dextrose and water or dextrose and saline solution) are given to replace the measured losses of the preceding 24-hour

period (12 hours for infants) together with additional fluid to compensate the insensible losses.

POSTOPERATIVE ELECTROLYTES

Sodium chloride is given in an amount of 6 Gm./L. of fluid lost by the gastrointestinal route during the preceding 24 hours. This may be administered as "half-normal" (0.45%) sodium chloride or as a "normal" (0.9%) solution in 5 or 10% dextrose. An additional 4.5 Gm. of sodium chloride may be given to compensate losses via urine and sweat. As a rule, all of the fluids prescribed for a patient need not contain electrolytes because a major segment of the 24-hour fluid losses (the insensible perspiration) does not contain electrolytes.

Exceptions to the foregoing are discussed on pp. 100-102.

Potassium should be added to the intravenous regimen when a patient must be maintained on intravenous fluids for more than 2 days. If there has been a preoperative potassium deficiency, potassium should be given postoperatively as soon as practicable. Potassium should ordinarily not be given intravenously if the urinary output is less than 400 ml. in a 24-hour period.

Forty to 50 mEq./day (approximately 4 Gm. KCl) is a satisfactory maintenance dose of potassium for adults if there are no fluid losses from the gastrointestinal tract. Additional amounts of potassium must be given to replace gastrointestinal losses. Approximately 20-25 mEq. of potassium should be given per day for each liter of gastrointestinal fluid which has been lost during the previous day. However, a total of more than 100 mEq. of potassium is seldom required during a single 24-hour period.

Sodium lactate (or sodium bicarbonate) solutions may be required instead of sodium chloride in replacement of fluid losses which predispose to acidosis (see table on p. 104). A useful guide for replacement of gastrointestinal losses is outlined below. This table* relates the proportions of the various fluids to use in replacement of gastrointestinal losses to the source of the loss.

Source of Loss	Dextrose in Water	Dextrose in Saline	M/6 Sodium Lactate
Gastric			
Average	33%	67%	
Low acidity	67%	33%	
Small intestine	20%	70%	10%
Ileostomy	10%	75%	15%
Bile		67%	33%
Pancreas		50%	50%

PARENTERAL SOLUTIONS
(See table on p. 116.)

Sodium chloride solution is the most commonly employed electrolyte solution. Although so-called "normal" (actually isotonic, 0.85-0.9%) sodium chloride is the most commonly used concentration, there is now a tendency to prefer "half-normal" (hypotonic, 0.45%) solutions with a view to administering the electrolyte over a longer period of time in order to promote better utilization.* It must be emphasized that the total amount of salt required must still be provided regardless of the concentration of sodium chloride in the solutions given.

Hypertonic saline solution (3-6% solutions of sodium chloride) is required in the treatment of acute water intoxication (see p. 109). It may also be given initially (followed by normal saline) in acute sodium depletion.

Sodium lactate is approximately isotonic in a $1/6$ molar (M/6) concentration. It is used to repair acidotic states. However, in order to be effective as an alkalinizing agent the lactate radical must be metabolized to bicarbonate, in severe acidosis, or in pathologic states where hepatic function is depressed (as may occur in prolonged hypotension or in shock), sodium bicarbonate may therefore be preferred. This is available in ampuls containing 3.75 Gm. (45 mEq.) in a 50 ml. volume. It is to be added to dextrose and water in the quantity required to provide the calculated need (see p. 118).

Lactated Ringer's injection resembles plasma in its electrolyte concentration. It differs from Ringer's injection by its content of lactate (bicarbonate precursor). The small amounts of potassium and calcium in these solutions are of insignificant value in therapy. The only advantage of lactated Ringer's injection over sodium chloride solutions is in the maintenance of patients who may develop acidosis. The so-called "balanced electrolyte" solutions which have recently come into use are somewhat more useful for this purpose because of their higher content of bicarbonate precursors (lactate, citrate, or acetate). Their content of potassium, however, may not be adequate. It is important to emphasize that balanced electrolyte solutions or lactated Ringer's injection should not be used in patients with alkalosis. These solutions are therefore not indicated to replace losses from the stomach or upper small intestine.

Darrow's solution is especially useful for replacement therapy in patients with acidosis and potassium deficiency. Thus it is particularly indicated for deficit therapy of diarrhea, especially in infants and children, and as a replacement fluid in the treatment of diabetic coma.

Ammonium chloride solutions should be used only for the initial treatment of very severe metabolic alkalosis. In most instances increased amounts of potassium chloride together with sodium chloride will satisfactorily correct metabolic alkalosis. Ammonium-containing solutions may be toxic, particularly in the presence of depressed liver function. They should be given slowly and with

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*The electrolyte-containing solutions are usually made up in at least 5% dextrose solution (isotonic) rather than in water. For this reason all of these solutions are actually hypertonic to varying degrees.

careful attention to development of the neurologic signs of ammonium intoxication.

Calcium gluconate may be required to treat tetany which accompanies uncompensated alkalosis or hypocalcemic states. The usual dose is 10 ml. of a 10% solution I.V., which may be repeated hourly as required for control of symptoms. In alkalosis, correction of the alkalotic state will promptly correct the tetany because the syndrome is in this instance actually attributable to the elevated blood pH rather than to an absolute calcium deficit.

A summary of the various electrolyte repair solutions described above is given in the table below. The solutions described in the table are the standard formulas generally available. However, it is a simple matter to prepare other types of electrolyte repair or maintenance solutions of varying constitution to suit special needs. This is conveniently accomplished by the addition (to 5 or 10% of dextrose in water) of electrolytes prepared in concentrated form in 20 or 40 ml. vials. Many manufacturers supply solutions of potassium chloride containing 20 or 40 mEq. of potassium in a 20 ml. vial, 50 mEq. NaCl in 20 ml. or 100 mEq. in 40 ml.; 50 mEq. of sodium lactate in 20 ml. or 100 mEq. in 40 ml., etc. By the use of these concentrates one can "tailor" solutions for more precise therapeutic purposes than can be served by the fixed formulations usually available. An example of the value of tailored solutions would be their use in the type of electrolyte therapy recommended in the table on p. 114. These special solutions are also of value in the treatment of patients with acute or chronic renal insufficiency, where the day-to-day needs for electrolytes may be highly variable.

Composition of Electrolyte Repair Solutions (in mEq./L.)

	Na^+	K^+	Ca^{++}	Cl^-	HCO_3^-*
Normal saline (0.85% NaCl)	145			145	
Half-normal saline (0.45% NaCl)	77			77	
Hypertonic saline (3% NaCl)	513			513	
1/6 Molar (M/6) sodium lactate	167				167
Lactated Ringer's Injection, U.S.P.	130	4	3	109	28
Ringer's Injection, U.S.P.	147	4	4	155	
Darrow's solution (KNL)	122	35		104	53
Balanced electrolyte solution†	140	10	5	103	55
Ammonium chloride (0.9%)‡				170	
Calcium gluconate (10%)			446		

*As bicarbonate or its precursors (such as lactate, citrate, or acetate).
†Contains also 3 mEq./L. of Mg^{++}.
‡Contains also 170 mEq./L. of NH_4^+.

CALCULATIONS USED FOR REPLACEMENT THERAPY
OF ELECTROLYTE DEFICITS

Although some idea of the quantity of electrolyte required to correct an electrolyte deficit is helpful, it must be emphasized that no method of calculation is so completely trustworthy that it can be recommended as a rigid prescription for replacement therapy. Clinical and biochemical response must be used as the primary guide; mathematical calculations are of value only as a preliminary basis for planning fluid and electrolyte therapy.

Sodium Chloride Deficit.

Total body water is used as the basis for calculation of sodium deficits because sodium is directly concerned with the movement of water in and out of cells, in order to correct deficits it may be necessary to supply sufficient osmotic activity to mobilize or retain large quantities of fluid. For the purpose of sodium replacement, 50% of body weight is taken to represent the clinical average of total body water.

Example A:
1. A 70 Kg. patient with a history of excessive sodium and chloride losses which have not been adequately replaced is found to have a serum sodium of 120 mEq./L.
2. The deficit of sodium, based on a normal of 140 mEq./L., is 140 − 120 = 20 mEq./L.
3. Total body water (50% of 70 Kg.) = 35 L.
4. Sodium deficit (20 × 35) = 700 mEq.
5. One L. of 0.9% sodium chloride contains approximately 150 mEq.
6. The total quantity of 0.9% NaCl theoretically required = 700 ÷ 150 = about 4.7 L. (4700 ml.).
7. Give **one-half** (2500 ml.) the calculated dose in 12-24 hours; if biochemical and clinical signs warrant, the remainder can be given in the next 24-48 hours. Remember that deficit therapy must be given in addition to the required maintenance therapy.

Example B: Assume that the patient in Example A has the same low value of serum sodium but that hyponatremia is not due to sodium chloride losses but to excessive water intake (dilutional hyponatremia; water intoxication; see p. 109). If the symptoms are mild, complete restriction of fluid until the serum sodium returns to normal may be all that is needed. However, in severe states of water intoxication, hypertonic solutions of sodium chloride are required. The solutions usually employed range in concentration from 3-6% NaCl. While hypertonic solutions are being used, rigidly restrict fluid intake only as necessary for administration of the hypertonic solution. The degree to which water must be restricted governs the choice of solution, i.e., 3% up to 6%. A 6% solution of NaCl contains approximately 1 mEq. of sodium/ml. Thus, if a 6% solution is used, the volume of solution to be used equals the quantity of sodium required. In

Example A, 700 ml. of a 6% solution would theoretically be required. Again it is to be emphasized that only one-half of the calculated dose would be given in the first 12 hours. After that the patient should be sufficiently improved to permit recalculation of his needs on the basis of another electrolyte determination.

Bicarbonate Deficit.

Use only the extracellular fluid space for calculation of the amount of alkali required. This will probably suggest a dose that is too low for complete correction; but the calculated dose, when given over a 24-hour period, should be adequate for the immediate emergency. A reappraisal of the biochemical and clinical status of the patient can then be made and additional therapy provided to restore the plasma bicarbonate to a normal level.

Example:

1. A 70 Kg. patient with a plasma bicarbonate of 15 mEq./L.
2. Normal plasma bicarbonate = 26 mEq./L.
3. Deficit = 26 − 15 = 11 mEq./L.
4. Extracellular fluid (20% of 70 Kg.) = 14 L.
5. Theoretical bicarbonate deficit = 11 × 14 = 154 mEq.
6. Administer 1 L. (166 mEq.) of M/6 sodium lactate in addition to maintenance therapy to make an initial correction of this bicarbonate deficit.

FLUID THERAPY IN CHILDREN

In children, a larger proportion of the total body water (50% of body weight) is in the extracellular space than is the case in adults (20% of body weight). Furthermore, the average daily output of fluid in an infant is equal to about one-half the extracellular fluid volume, whereas in the adult the output is only about one-seventh the extracellular volume. Because of this rapid turnover of fluid, the infant is able to tolerate only very short periods of negative fluid balance before severe dehydration results. The renal regulation of electrolyte and acid-base balance is also less efficient in the infant than in the older child or adult.

ESTIMATION OF FLUID AND ELECTROLYTE REQUIREMENTS FOR MAINTENANCE IN CHILDREN

The fluid and electrolyte needs of infants and children vary with the age of the child. It is therefore usual to express these requirements on the basis of body weight.

Average requirements for water, electrolyte, and carbohydrate in infants and children are as follows (see also the table on p. 100):

	Water (ml./Kg.)	Sodium (mEq./Kg.)	Potassium (mEq./Kg.)	Dextrose (Gm./Kg.)
Premature	50	1.5	1.5	2
Newborn	40	0.5	0.5	2
4- 10 Kg.	120- 140	1.8- 1.5	1.8- 1.5	5
10- 20 Kg.	100- 80	1.5- 1.3	1.5- 1.3	4
20- 40 Kg.	80- 60	1.3- 1.0	1.3- 1.0	3

Except in premature infants, a solution containing 15 mEq./L. each of sodium, potassium, chloride, and bicarbonate (or bicarbonate precursor, as lactate) is convenient to provide the maintenance requirements listed above. To 900 ml. of 5% dextrose in water, add 90 ml. M/6 sodium lactate (or 16.7 ml. of 7.9% sodium bicarbonate) and 7.5 ml. of 14.9% potassium chloride. The solution may be administered in a quantity determined by the maintenance water requirement shown above. It is desirable that the required fluids and electrolytes be given slowly over the greater part of the 24-hour period for which they are calculated.

The maintenance requirements listed above may be reduced in the immediate postoperative period or in edematous states. They may be increased if activity exceeds quiet bed rest and in conditions characterized by salt loss, such as adrenal insufficiency or salt-losing nephritis, as well as in hyperventilation, fever, and excessive sweating. In fever, increase the requirements 8% per degree Fahrenheit or 12% per degree centigrade. The precautions against administration of potassium in the presence of a reduced urinary output must also be observed, if preoperative potassium deficiency has been corrected, it is not necessary to give potassium until the second postoperative day.

CORRECTION OF FLUID AND ELECTROLYTE DEFICITS

When water and salt deficits have been incurred it is necessary to give fluid and electrolyte in addition to that required for maintenance. The usual deficits which may be incurred in severe dehydration (after acute loss of 12-15% body weight and with clinically evident signs of dehydration) are shown below.

	Water (ml./Kg.)	Sodium (mEq./Kg.)	Chloride (mEq./Kg.)	Potassium (mEq./Kg.)
Diarrhea	100- 150	8- 12	7- 10	8- 14
Pyloric stenosis	150- 200	12- 17	15- 19	10- 14
Diabetic acidosis	100- 150	6- 12	5- 9	4- 7

In mild dehydration (approximately 5% loss in body weight), reduce the above intakes proportionately.

If extrarenal losses persist it is necessary to continue to give fluid and electrolyte in addition to maintenance requirements. The nature of the electrolyte losses by way of the gastrointestinal tract

may be recalled by reference to the table on p. 104. A frequent cause of fluid and electrolyte losses in infants is by way of diarrheal stools. With severe diarrhea the fluid loss approximates 50-70 ml./Kg./day; in less severe cases, 20-40 ml./Kg./day. The electrolyte losses are (in severe cases):

Sodium	3-6 mEq./Kg./day
Potassium	3-5 mEq./Kg./day
Chloride	2-4 mEq./Kg./day

FLUID AND ELECTROLYTE THERAPY IN ACUTE RENAL INSUFFICIENCY

In the oliguria of acute renal insufficiency which may occur as a result of trauma, shock, transfusion reactions, or tissue insult from various causes (so-called lower nephron nephrosis, lower nephron syndrome, etc.) it is important to restrict fluid and electrolyte intake to the minimum requirement. For this reason this syndrome must be diagnosed promptly so that excess administration of fluid and electrolyte - which may easily produce generalized edema and death from respiratory and cardiac failure - can be avoided.

Except for the first few hours after operation, acute renal insufficiency should be suspected in any patient previously well hydrated who exhibits (1) oliguria (less than 400 ml. of urine/day*) in the presence of a normal BP, and (2) urine of low sp. gr. (1.008-1.014).

The fluid intake of the oliguric patient must be strictly limited to that lost by all routes. Ordinarily this will total no more than 500-800 ml./day (12 ml./Kg. for insensible losses plus the output in the urine for the previous 24 hours less 250 ml. produced endogenously as metabolic water of oxidation). If visible perspiration occurs or if there are gastrointestinal losses, as in the presence of diarrhea, vomiting, or gastric suction, these fluid losses must also be replaced. Unless the oliguric patient loses about 0.5-1 lb. of body weight/day he is probably being overhydrated.

Because of tissue breakdown, potassium is liberated in increased quantities. Since potassium cannot under these circumstances be excreted, it may rise to toxic levels in the blood. The accumulation of potassium in the extracellular fluid is perhaps the most serious complication of oliguria. In an attempt to spare tissue breakdown and thus minimize the mobilization of potassium and nitrogenous waste products such as urea, hypertonic (25-50%) solutions of dextrose are used. These must be given slowly with a view to maintaining elevated levels of the blood sugar throughout the 24 hour period. The addition of insulin to hypertonic dextrose solutions aids in the storage of glycogen in the tissues, and this process tends to shift potassium from the extracellular fluid. For this purpose 1 unit of regular insulin may be added for each 2 Gm. of dextrose administered (50 units in 500 ml. of 20% dextrose).

*It is not usual for absolute anuria to occur in these patients. If this does occur, obstructive uropathy should be strongly suspected.

The accumulation of phosphate in the oliguric patient leads to a depression in the blood calcium, which enhances the cardiac toxicity of the elevated serum potassium. Calcium is therefore administered in doses of 50-100 ml. of 10% calcium gluconate added to 500 ml. of the hypertonic dextrose solutions to antagonize the elevation in potassium.

It is obvious with the very restricted electrolyte and fluid intake permitted these patients that complete correction of electrolyte and acid-base imbalance is not possible. However, normal saline and/or M/6 sodium lactate may be used in conservative amounts (250-400 ml./day).

Potassium intoxication may be treated by the use of a sulfonated polystyrene resin in the sodium cycle such as Kayexalate®. The resin may be given by mouth every 4-6 hours, the average dose being 15 Gm. suspended in a minimal amount of fluid. When the resin is being administered orally, a mild laxative should also be given to guard against intestinal impaction. The dose range and duration of therapy vary widely depending on the status of the serum potassium and the Ecg. Frequently the oral route cannot be used because of nausea and vomiting. In such cases the resin can be given every 4 hours by rectum as a retention enema. It is desirable that the resin be retained for about 3 hours and then washed out. This resin is more effective in removing potassium than those previously used for this purpose. Consequently, the effect of therapy with resin on the electrolyte changes in the patient must be carefully appraised at regular intervals.

When urine flow is restored, renal tubular function is still poor, so that disorders of fluid and electrolyte regulation are to be expected. During this so-called "diuretic" phase very careful attention to and the prompt replacement of electrolyte losses is of utmost importance until recovery of renal tubular function is assured.

In severe cases of acute renal insufficiency not amenable to the conservative therapy described above, extracorporeal dialysis may be required. Either the Kolff-Merrill apparatus or the Travenol coil kidney are of great value in such cases. If these are not available, intermittent peritoneal lavage may be indicated. A simplified procedure and the indications for the use of this technic have been described by Doolan & others (Am. J. Med. 26:831, 1959). Where it is applicable, peritoneal lavage can be attempted in any hospital because it requires no special equipment or dialyzing solutions that are not readily available.

ROUTES OF PARENTERAL FLUID ADMINISTRATION

VENOCLYSIS

Solutions containing glucose, electrolytes, or amino acids, as well as fat emulsions, and whole blood or plasma fractions, may all be administered intravenously. Fluids which are isotonic and of a neutral pH are tolerated best. Pure water must never be given

intravenously because it will cause hemolysis. When electrolyte-free solutions are required in therapy, solutions of 5-10% dextrose in water are used. Solutions of sugars which are more hypertonic than that of a 10% glucose solution produce phlebitis and obliteration of veins when administered for other than a very short period. If hypertonic solutions are necessary, as may be the case in patients who require an increased caloric intake in a small volume (such as in acute renal insufficiency; see p. 43), the infusions should be made through a polyethylene cannula threaded high into a large vein with a free flow of blood (see below).

Sites for Venoclysis.

The veins of the forearm and of the back of the hand are preferred. Infusions into the antecubital vein have the disadvantage of requiring prolonged extension of the elbow. The long saphenous vein in the leg and its tributaries on the dorsum of the foot are convenient, but occasionally a deep femoral phlebitis results. Femoral vein puncture about 3 cm. below the inguinal ligament, using the femoral artery (which lies just lateral to the vein) as a landmark, may be necessary in emergencies when safer and more superficial veins are temporarily inaccessible. The scalp veins of infants can be punctured with a No. 24-21 gauge needle. The superior longitudinal sinus should not be used.

Venous Cut-down.

When prolonged, continuous infusion is needed, when venipuncture is difficult, when a secure infusion is essential, when rapid infusion is important, or when emergency situations combine these indications, it is advisable to "cut down" on a vein in order to tie a cannula or polyethylene tube in place. The distal saphenous vein, lying just anteromedial to the medial malleolus of the tibia, is first choice. When repeated cut-downs are necessary, this vein (which has a fairly predictable course) may be exposed and cannulated at successively higher levels between the ankle and groin. Alternatively, the median cubital or the basilic vein of the antecubital region (there are many variations of venous anatomy here) or any other sizable superficial vein may be chosen.

Rate of Administration.

Isotonic and hypotonic solutions may usually be given intravenously at a rate of 250-500 ml./hour. When glucose is present in the infusion, the rate of administration should not exceed 0.5-0.75 Gm. of glucose/Kg. body weight/hour to avoid glycosuria and diuresis (e. g., in a 70 Kg. patient, 1000 ml. of 5% glucose solution should take at least 90-120 minutes to administer). Potassium should usually not be administered faster than 20 mEq./hour.

HYPODERMOCLYSIS

This procedure consists of the subcutaneous administration of isotonic fluid, usually by gravity flow from a reservoir. In surgical patients, the chief indication for hypodermoclysis is inability to find a vein for venoclysis. In small infants this occurs frequently; in adults it occurs very rarely.

Only fluids which are approximately isotonic should be given subcutaneously. Glucose, 2.5%, in 0.45% saline is a satisfactory solution, or physiologic saline or Ringer's injection may be given. Glucose, 5% in water, is acceptable but causes more local reaction and is definitely contraindicated in debilitated elderly patients.

In adults the subcutaneous tissues of the anterior or lateral aspect of the mid thigh are preferred. In small infants, fluid can also be injected subcutaneously in the upper back, axillae, and lower abdomen, using a 20-50 ml. syringe and No. 20 gauge needle.

In infants, up to 50 ml. can be injected into each of several sites at 1 time to a total volume of up to 30-40 ml./Kg. body weight. In adults, 750 ml. can be injected into each thigh at 1 time at a rate of 250-500 ml./hour for each thigh. The practical maximum which can be absorbed in 1 day is 3000-4000 ml. Hyaluronidase (250 viscosity units/500-1000 ml. of fluid) will increase the absorption rate about 12 times if it is injected into the tubing at the beginning of the clysis, or into the solution. Hyaluronidase is contraindicated with solutions containing large amounts of potassium, as it may produce excessively rapid absorption. Two ml. of 1% procaine added to each 1500 ml. of solution lessens the discomfort of the clysis.

Precautions.
   A. Never give concentrations of glucose greater than 5% to any patient, as sloughing of tissues may occur.
   B. Avoid over-distention of tissues because it will cause avascularity and necrosis. Whenever the skin over a subcutaneous clysis becomes blanched, discontinue the clysis temporarily or select another site.
   C. Rigid aseptic technic is necessary to avoid cellulitis and abscess formation.

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Surgical Infections *

STERILIZATION

The only completely reliable methods of sterilization are by steam under pressure (autoclaving) and by dry heat.

Autoclaving.
Saturated steam at a pressure of 750 mm. Hg (15 lb./sq. in. above atmospheric pressure) at a temperature of 121°C. (250°F.) destroys all vegetative bacteria and most resistant dry spores in 13 minutes. Additional time (usually a total of 30 minutes) must be allowed for the penetration of heat and moisture into the center of packages.

Dry Heat.
Exposure to continuous dry heat at 160°C. (320° F.) for 1 hour will sterilize articles which would be spoiled by moist heat or which are more conveniently kept dry If grease or oil is present on instruments, safe sterilization calls for 4 hours' exposure at 160°C. (320°F.).

Boiling.
Instruments should be boiled only if autoclaving and dry heat are not available. The minimum period for sterilization in boiling water is 30 minutes at altitudes less than 300 meters (90 feet). At higher altitudes the period of sterilization must be increased. The addition of alkali to the sterilizer increases bactericidal efficiency by raising pH so that sterilization time can safely be decreased to 15 minutes.

Soaking in Antiseptics.
In general, sterilization by soaking should never be relied upon if autoclaving or dry heat is suitable and available. Certain cutting instruments, however, require soaking. A commercially available noncorrosive preparation for this purpose is Bard-Parker Formaldehyde Germicide® containing hexachlorophene:

    ℞ Solution of Formaldehyde, U.S.P.   130
        Potassium nitrite   0.15
        Sodium hydroxide   0.012
        Hexachlorophene   5
        Ethyl alcohol (C.P. 95%), q.s. ad 1000

Plastic materials and instruments containing shellac, lenses, or electric connections are best sterilized by soaking in aqueous benzalkonium germicide:

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*Anti-infective drugs and dosages are listed in the Appendix, pp. 614-621. Rabies is discussed on p. 33.

℞ Benzalkonium chloride concentrate,

12.5% by weight	10
Sodium nitrite	2
Distilled water, q. s. ad	1000

These solutions will destroy vegetative bacteria in 30 minutes, but soaking for at least 18 hours is required to destroy spores.

ANTISEPTICS

Antiseptics are chemical agents which kill bacteria or arrest their growth; they may or may not be sporicidal. Antiseptics should not be used in fresh wounds since their toxicity to host cells far outweighs the possible advantage of their antibacterial effect. Many new and more versatile antiseptics have been developed in recent years. The most promising of these are discussed below.

Antiseptics for General Utility Purposes.

Soap solution is 1 of the best all-purpose cleansing agents, but it is a weak antiseptic. The following preparations are more bactericidal than soap and are especially useful in the hospital or office for cleaning floors, furniture, and operating room equipment and for soaking contaminated utensils and materials. They are preferred in the order listed below:

A. Phenol Compounds: Phenol (carbolic acid) is 1 of the most potent bactericidal chemicals available, but is too caustic for safe use. The same is true to a lesser degree of cresol (Lysol®). These have been superseded by such alkyl and aryl phenol germicides as Amphyl®, which can be used effectively in 2% solution on furniture and inanimate surfaces and in 0.5% solution as an antiseptic handwash. Amphyl® kills bacteria, fungi, and tubercle bacilli, and remains active on the surface for 7 days or longer. Staphene® and San-pheno-X® are similar products.

B. Iodophors: These are chemicals in which iodine is combined with a detergent (Wescodyne®, Virac®), or with polyvinylpyrrolidone (Betadine®, Isodine®). The toxicity of the iodine is practically eliminated, but surface and skin disinfection is efficient.

C. Alcohols: 70% ethyl alcohol is a powerful germicide. The dilution of isopropyl alcohol is apparently less critical; 30-50% solutions are also potent.

D. Quaternary Ammonium Compounds: These are less effective than phenol, iodine, and alcohol and are inactivated by soap. Benzalkonium Chloride, U.S.P. (Zephiran®, Roccal®), is used in 1:750 or 1:1000 aqueous solution for general disinfection or for soaking instruments (see above).

E. Other Chemicals: Formaldehyde, hypochlorites, chloramine, mercury salts, and solutions of azo dyes have been largely replaced by more reliable compounds.

Skin Antiseptics.

The most important applications of skin antisepsis are the hand scrub of the operating team and the preparation of the operative

field. Hexachlorophene is widely used in skin disinfection, usually in combination with a detergent (e.g., pHisoHex®) or with liquid or bar soap (e.g., Septisol®, Gamophen®, Dial®). Daily use of hexachlorophene produces a sustained lowering of the bacterial count on the skin. Alcohol dissolves hexachlorophene and should not be applied if a prolonged surface effect is desired.

A. Hand Scrub Routine: Always scrub for 10 minutes except when changing gloves and gown aseptically between clean cases; in these circumstances scrub for 5 minutes.
   1. Wash hands and forearms thoroughly for 30 seconds with a hexachlorophene preparation.
   2. Clean fingernails.
   3. Scrub for 5 minutes with a sterile brush, covering the entire surface of the hands and forearms repeatedly with the hexachlorophene preparation.
   4. Scrub for another 5 minutes with a second sterile brush (a total of 10 minutes by the clock).

B. Preparation of the Operative Field:
   1. On the day before the operation - Shave the operative field and wash the skin thoroughly for 10 minutes with soap, detergent, or a hexachlorophene preparation. Clean crevices with a cotton swab. In the preparation of patients with unclean habits it may be necessary to scrub such areas as the groin or feet on several successive days before surgery.
   2. In the operating room - Use aseptic technic and prepare as follows:
      a. Wash the operative field for 10 minutes with gauze and a detergent or soap solution. Rinse with water and dry with a towel or gauze.
      b. Apply a disinfectant in either of the following ways -
         (1) Apply an iodophor (see p. 125) to the skin or swab the surface of the skin for 1 minute with a tincture (e.g., tincture of benzalkonium). Allow to dry before draping.
         (2) Apply Tincture of Iodine, U.S.P., 2%, allow to dry, and remove with 5 applications of 70% alcohol. Inorganic iodine is 1 of the most efficient skin antiseptics available, but it occasionally causes skin reactions. Do not use iodine on the perineum, genitalia, or face; on irritated or delicate skin (e.g., small children); or when the patient gives a history of iodine sensitivity.

SPECIFIC TYPES OF SURGICAL INFECTIONS

FURUNCLE

A furuncle or boil is an abscess of a sweat gland or hair follicle, usually caused by Staphylococcus aureus. Treatment consists of hot packs and, if necessary, incision and drainage when the process has localized. Squeezing or incision before localization is to be avoided. Antibiotics are rarely indicated. Furuncles are usually

self-limited and of minor significance except when multiple (furunculosis) or when they occur on the face. Rule out diabetes by urinalysis in every case of multiple or recurrent furuncles.

Furunculosis.

Predisposing factors include low resistance to infection, as in uncontrolled diabetes or debilitation; high virulence of the invading staphylococcus; poor body hygiene; and folliculitis. Treatment consists first of controlling diabetes or other systemic or local predisposing conditions. Have the patient bathe all over once or twice daily with soap containing hexachlorophene. Use aseptic dressing technic on draining furuncles to avoid contaminating surrounding skin. Administration of an antibiotic chosen by sensitivity studies may occasionally be necessary. Autogenous vaccine may have some value in resistant cases.

Furuncles of the Face.

Infections in the mastoid and occipital areas and on the face above the level of the mouth may produce septic phlebitis of the emissary veins with extension into the cavernous sinus. Furuncles in these regions are therefore dangerous and must not be squeezed or incised. They should be allowed to localize under treatment by rest and hot packs. Drainage can then be encouraged by gentle manipulation. Antibiotics chosen by sensitivity tests are frequently advisable.

CARBUNCLE

Carbuncle is a staphylococcal infection of the skin and subcutaneous tissues which resembles a cluster of partially confluent furuncles. There is considerable surrounding brawny induration, and localization tends to proceed slowly. The back of the neck is a common site. Fever, malaise, and prostration may occur. Carbuncles are more common in diabetic patients, and diabetes should be ruled out in every case. These lesions can be quite extensive locally, and may endanger life in debilitated or diabetic patients.

Treatment.
  A. Antibiotic therapy will frequently lead to subsidence of the process or reduce the extent of surgery required.
  B. General supportive measures include application of hot moist packs to the lesion, control of diabetes if present, rest, and symptomatic treatment of malaise and pain. •
  C. Incision and drainage or excision of the area is necessary for patients who fail to respond to antibiotics and supportive measures.

CELLULITIS, LYMPHANGITIS, AND LYMPHADENITIS

These common, nonsuppurative infections of the subcutaneous tissues may be caused by aerobic or anaerobic bacteria. The usual sequence of events is the development of an area of spreading infection and cellulitis, generally at the site of an infected wound.

Involvement of the lymphatic channels is indicated by the red
streaks on the skin extending proximally to draining nodes - which
are usually, by this time, tender and enlarged. Rarely the nodes
will suppurate and form abscesses requiring drainage. General
measures for this group of infections include rest or splinting, ele-
vation, hot packs, drainage of the entry wound if necessary, and
symptomatic treatment of the systemic reaction, which usually
consists of moderate fever and malaise. Antibiotic therapy is in-
dicated when cellulitis is extensive or rapidly progressive, and
should always be instituted when lymphangitis or lymphadenitis
appears. Penicillin should be given when cultures for sensitivity
studies cannot be obtained since the hemolytic streptococcus is
frequently the cause.

The hemolytic streptococcus is also responsible for the follow-
ing 2 severe types of cellulitis:

A. Erysipelas: This form of cellulitis is characterized by a
   marked erythematous blush of the skin in and around the area
   of infection. Elderly or debilitated patients are more suscep-
   tible. Penicillin is specific. General measures are as out-
   lined in the preceding paragraph.

B. Hemolytic Streptococcus Gangrene: This is a rare and intense
   form of streptococcal cellulitis which progresses to necrosis
   of the skin and subcutaneous tissues. It is most likely to occur
   if the blood supply to the part is diminished, as in peripheral
   vascular disease of an extremity. Gangrene may be prevented
   if the virulent nature of the infection is recognized early, when
   it begins to involve the fascial planes. The fascia should then
   be opened widely to allow free drainage. Large doses of peni-
   cillin and general measures as above are recommended.

PILONIDAL SINUS

Pilonidal sinus or cyst is caused by the penetration of hair into
the subcutaneous tissues as a result of trauma. The hair acts as
a foreign body and nidus for the development of infection. The com-
monest location is in the midline of the back over the sacrococcygeal
junction, but the lesion may also occur on the hands or in the um-
bilicus. The majority of patients are hirsute males in their late
teens or early twenties. The first symptom is either an acute ab-
scess or a chronic draining sinus. Sinus tracts may ramify to form
several openings on either side of the midline. When a sinus open-
ing is near the anus it may be confused with fistula in ano.

Treatment.

Treatment of the acute abscess is by incision and drainage,
usually under local anesthesia. If the infected cavity or "cyst" is
small and superficial, unroofing the abscess and packing it open,
followed by a sitz bath daily, may result in complete healing. As
a rule, however, a chronic draining sinus persists which must be
surgically extirpated by excision and primary closure or, prefer-
ably, by an "open" technic such as marsupialization. Marsupial-
ization can be done under local anesthesia in the outpatient clinic
with minimum morbidity. The procedure consists of opening the
sinus and all of its ramifications over a grooved director or

hemostat. The sinus tract is then completely exteriorized by cir-
cumcising the overhanging skin edges. Finally, the intact skin
edge is sutured to the edge of the sinus tract and a pressure dress-
ing applied. There is little postoperative pain. The pressure
dressing is removed on the second postoperative day. Sutures are
removed in 7-9 days and daily sitz baths are then begun. The
wound is inspected every 5-7 days and wiped out carefully to pre-
vent bridging-over. Healing requires an average of 3 weeks. Daily
dressings are applied by the patient, who is entirely ambulatory
throughout the treatment and is not hospitalized. The recurrence
rate with this method is 5-10%.

PROGRESSIVE BACTERIAL SYNERGISTIC GANGRENE

This is a mixed and synergistic infection with a microaerophilic
nonhemolytic streptococcus (derived from the patient's intestinal
tract) and hemolytic, coagulase-positive Staphylococcus aureus.
Most cases involve the abdominal wall. The lesion may develop
following drainage of a peritoneal abscess or an empyema, wound
infection, or infection around an ileostomy or colostomy.

The principal symptom is excruciating pain and tenderness in
the infected area. The lesion consists of progressive superficial
ulceration and a narrow circle of gangrenous skin surrounded by a
margin of erythema. The diagnosis is suggested by the course and
appearance of the infection, which may progress to involve large
areas of the abdominal wall, flank, and neighboring regions. Care-
ful bacteriologic methods are required to identify the microaero-
philic streptococcus which grows in the advancing margin of the
ulcer.

Treatment.

Appropriate antibiotic therapy is indicated, usually following
cultures and sensitivity studies. In some cases penicillin will be
effective. Bacitracin has proved to be among the most valuable
agents in this type of infection, and is used both parenterally and
topically. If chemotherapy is not effective it may be necessary to
excise the region completely (including the erythematous margin).
Sterilized medicinal grade zinc peroxide, freshly prepared by sus-
pension in distilled water to make a thin, creamy paste, is then
applied once or twice daily to the wound and sealed over with vase-
line gauze in order to prevent recurrence of infection. Skin grafting
is required after the infection has been controlled.

ANTHRAX

Anthrax is an acute infectious disease of domesticated animals
(cattle, horses, sheep, swine) which may be transmitted to man
either directly or by contact with hides or hair. The characteristic
lesion in humans is a "malignant pustule" resembling a carbuncle
but distinguished by a central necrotic eschar surrounded by viola-
ceous, bullous cellulitis. The diagnosis is made by identifying the
gram-positive anthrax bacilli in the serum from 1 of the blebs.

Treatment is with large doses of penicillin. Surgical intervention
is not necessary for the acute process.

TETANUS

Tetanus is a nervous system intoxication caused by the toxin
of Clostridium tetani. The organism most often infects puncture
wounds or injuries associated with devitalized tissue (anaerobiosis),
such as compound fractures, but even trivial wounds may be infec-
ted.

Clinical Findings.
The incubation period is usually 7-14 days, but symptoms can
appear as early as 24 hours or as long as 8 months after injury.
The first symptom may be pain and tingling at the site of the con-
taminated wound. Tetany may be limited to the injured extremity,
or local spasm may be the first sign of the disease. More fre-
quently, however, the earliest evidence of nervous system irritation
is trismus ("lockjaw") followed by spasms which gradually pro-
gress to involve the rest of the body. Dysphagia, opisthotonos, a
rigid but nontender abdomen, spasms of the facial muscles (risus
sardonicus), and laryngeal spasm are classical manifestations.
Trivial stimuli such as bright illumination or jarring the bed may
elicit gross spasms or convulsions.

Death is most frequently caused by asphyxia from prolonged
spasm of the respiratory muscles. Pneumonia is also a common
cause of death.

The diagnosis of tetanus must be based on the clinical picture,
since the organism is difficult or impossible to recover.

Prophylaxis.
  A. Active Immunization: Three 0.5 ml. I.M. injections of tetanus
     toxoid (APT) at intervals of 3-6 weeks confer active immunity.
     A booster should be given every 4-5 years and at the time of
     injury.
  B. Passive Immunization: Tetanus antitoxin (TAT) is indicated
     for any wound potentially contaminated with tetanus bacilli in
     a patient who has not been actively immunized. Patients with
     dirty puncture wounds, compound fractures, major burns, and
     severe wounds should receive TAT. The administration of
     TAT in minor lacerations is a matter of judgment. When there
     is the slightest chance of contamination with tetanus, the pa-
     tient should be immunized. The usual dose of TAT is 3000
     units. Always test for sensitivity, and desensitize if necessary
     (see p. 13).
  C. Wound Care: Adequate debridement and delayed closure of
     wounds which are heavily contaminated and traumatized re-
     moves the conditions which favor the growth of tetanus bacilli.

Treatment.
Tetanus is a true emergency. Intensive treatment should be
started as soon as the earliest signs are noted. Ideally, the patient
should be under the combined care of an internist, a surgeon, and
an anesthesiologist.

A. Neutralization of Toxin: Toxin which has already entered and damaged the patient's cells is termed "fixed toxin," and cannot be reached by antitoxin. Circulating toxin must be neutralized with massive doses of antitoxin (TAT) if the preliminary skin test is negative. If the skin test is positive, rapid desensitization must be carried out (see p. 13).

  1. Systemic serotherapy - Give TAT, 50,000 units I.V. and 50,000 units I.M. Stat., followed by daily injections of 5000 units I.M. until the disease is under control. In critically ill patients it may be advisable to give up to 1 million units I.V. in 24 hours.

  2. Local serotherapy - Inject a total of 10,000 units of TAT into the tissues at multiple sites around the wound 1 hour or more before surgical treatment.

B. Surgery: Locally excise (or incise) and debride the suspected wound under local procaine or intravenous thiopental (Thiopental®) anesthesia when feasible. Withhold surgery for at least 1 hour after systemic serotherapy. The wound should be left open, and may be packed with zinc peroxide paste.

C. Control of Muscle Spasms: The patient should be placed in a dark, quiet room and should be spared all unnecessary movement, noise, and excitement. Muscle spasms and convulsions can be further minimized by sedatives and muscle relaxants (see pp. 603 and 624).

D. General Measures:

  1. Nursing care must be expert and constant.

  2. Fluid balance and nutrition (see p. 99) must be maintained by parenteral or nasogastric tube feeding.

  3. Tracheostomy (see p. 166) - A tracheostomy set should be at the patient's bedside. If respiratory arrest occurs or if excessive mucus blocks the airway, tracheostomy may be lifesaving. Tracheostomy should probably be performed on every severe case.

  4. Artificial respiration - Use of a mechanical respirator may be required for days or weeks.

  5. Antibiotics apparently do not influence the course of established tetanus but may be advisable to control pneumonia or secondary wound sepsis.

Prognosis.

The prognosis varies with the facilities available for treatment. The mortality rate in large series is 25-35%. The longer the incubation period, the better the outlook. If the incubation period is less than 7-10 days, the mortality rate will be almost twice as high as with longer incubation periods.

GAS GANGRENE

Gas gangrene, caused by Clostridium perfringens (welchii) and other toxigenic anaerobic clostridia, is an occasional complication of extensively traumatized and soil-contaminated wounds.

Clinical Findings.

The diagnosis is made primarily on clinical grounds. Do not wait for bacteriologic studies before starting therapy.

The onset is generally abrupt, 1-4 days after injury, although the first symptoms may appear as early as 6 hours after injury. Wound pain is the earliest symptom, followed soon by a rapid, feeble pulse, pallor, prostration, and, frequently, septic shock. Varying degrees of fever and leukocytosis are reported. Anemia usually appears rapidly as a result of extensive hemolysis. The wound becomes swollen and exudes a dirty brown, watery, sometimes malodorous discharge. Exposed muscle is usually discolored, edematous, and nonviable. Gross crepitation around the wound (caused by gas in the tissues) occurs sooner or later in most cases, and the gas can be demonstrated by x-ray before it becomes palpable. Spread of the infection along fascial planes and muscle bellies is rapid, and toxicity becomes overwhelming, sometimes within a few hours.

Prophylaxis.
- A. Debridement must be adequate to remove devitalized tissue; heavily traumatized and contaminated wounds should be left open.
- B. Antibiotic therapy with penicillin and the tetracyclines for contaminated wounds.
- C. Antitoxin is of no value in prophylaxis.

Treatment.

Gas gangrene is a surgical emergency. Begin therapy immediately.
- A. Antibiotics: Administer 1 million units of penicillin I.V. every 3 hours and tetracycline, 500 mg. I.V. every 4-6 hours, before and after operative treatment. Neomycin is also effective, but its usefulness is limited by its marked toxicity.
- B. Antitoxin: After appropriate skin tests and desensitization as necessary, give pentavalent gas gangrene antitoxin I.V. prior to surgery. The dose is 2-4 vials (about 20,000 units/vial). If the toxemia is not significantly improved within 4-6 hours, repeat the initial dose of antitoxin.
- C. Operative Treatment: General anesthesia, rapid and wide removal of affected tissue, and, frequently, excision of entire muscle bellies are necessary. The fascial planes and muscle sheaths must be freely incised to promote drainage. Pack the wounds loosely to keep them open, and splint extremities as required.
- D. General Supportive Measures: Combat septic shock (see p. 11), anemia, and fluid and electrolyte abnormalities.

Prognosis.

Gas gangrene can be an extremely fulminant disease, fatal within a few hours. The prognosis appears to be considerably improved when antiserum is used. In a series of 42 cases treated in a civilian hospital, Altemeier and others (A.M.A.Arch.Surg. 74:839, 1957) reported a mortality of 31% in patients treated with antitoxin compared with 50% for those who did not receive antitoxin.

ACTINOMYCOSIS

Actinomycosis is a chronic suppurative and granulomatous process caused by infection with Actinomyces israelii (A. bovis), a relatively common saprophytic resident of the mouth and the respiratory and gastrointestinal tracts. Invasion occurs through a break in the mucous membranes caused by infection or trauma. The 3 regions most commonly involved are the cervicofacial area (60%), the thorax (20%), and the abdomen (20%).

Clinical Findings.

There is usually a precipitating trauma or infection (e.g., dental extraction, lung abscess, appendicitis, or gastrointestinal perforation), followed by an indolent, burrowing network of sinuses and abscesses which tends to cross tissue planes indiscriminately and reach the body surface. Around the sinuses there is firm induration, and the overlying skin becomes brawny and dusky red. The granulation tissue is vascular, and drainage varies from serous to purulent. The systemic reaction is at first minimal, but as the infection becomes extensive the condition of the patient deteriorates from the effects of chronic sepsis. Pyogenic or intestinal organisms are invariably present also (mixed infection).

The diagnosis is made by finding "sulfur granules" in the drainage or curettings from sinuses and abscesses. These are tiny yellowish masses composed of a central dense network of mycelial filaments which project radially and are usually enveloped by eosin-staining hyalin clubs. The granules are often difficult to find, and the diagnosis of actinomycosis is thus not easy to establish. Repeated smears and cultures should be made and biopsy material obtained for pathologic examination.

Treatment.
  A. Medical Measures Perform sensitivity tests on culture material. Penicillin is usually the drug of choice for Actinomyces israelii infections. Dosage should not be less than 1 million units daily for 4-6 weeks. Massive doses (10-20 million units daily for a total of 500 million units) have proved more effective in some cases. Concomitant use of sulfadiazine may be of value. Bacitracin is also known to be useful. The selection of another agent to be combined with penicillin will depend on the nature of the mixed infection as determined by culture and sensitivity studies. Iodides and irradiation are of no value.
  B. Surgical Treatment: Radical drainage and excisional procedures are no longer necessary since the antibiotics have come into wide use. Sinuses and abscesses should be conservatively incised and drained as required by the mixed infection and as an adjunct to intensive and prolonged chemotherapy. In rare cases (e.g., tubo-ovarian actinomycotic abscess), resection of the infected focus may be advisable.

BLASTOMYCOSIS

Blastomyces dermatitidis infection is characterized by chronic ulcerating lesions of the skin, resembling boils with multiple draining sinuses; and pulmonary lesions which may simulate tuberculosis. Septicemic spread may follow pulmonary infection, and further abscesses may then develop in bone, brain, or subcutaneous tissues. Blastomyces brasiliensis causes primary papillomatous granulomas of the buccal mucosa. Involvement of the skin, lymphatic tissues, and viscera also occurs. The diagnosis of either variety depends upon microscopic recognition of the fungus in tissues and exudates, followed by its isolation in culture.

The treatment of choice is with amphotericin B.

HOSPITAL STAPHYLOCOCCAL INFECTIONS

Hospital cross-infection with hemolytic, coagulase-positive Staphylococcus aureus has become a serious problem within recent years. The strains of staphylococcus now endemic in many hospitals are resistant to penicillin, streptomycin, and the common broad-spectrum antibiotics (especially the tetracyclines), presumably as a consequence of the widespread use of these agents. Relaxation of aseptic precautions in the operating theater and wards and an unwarranted reliance on "prophylactic" antibiotics has further contributed to the development of resistant strains. The result is a significant increase in the incidence of hospital-acquired staphylococcal wound infection, pneumonitis, and septicemia, the latter 2 complications especially affecting infants, the aged, and the debilitated.

Preventive Measures.
 A. Hospital Administration:
    1. The surgical infection control program should be coordinated closely with that of other services through a Hospital Infection Committee set up to promulgate and enforce regulations.
    2. All staphylococcal infections should be reported immediately. A clean wound infection rate of more than 1-2% indicates a need for investigation.
 B. Cultures:
    1. Obtain culture and antibiotic sensitivity studies on wounds, ulcerations, and significant infections of all types.
    2. Phage-typing of staphylococci may be useful in studying epidemics.
 C. Isolation:
    1. Isolate every patient with a staphylococcal infection or draining wound.
    2. Isolate every case of suspected staphylococcal infection until the diagnosis has been ruled out.
    3. Isolate every patient in whom staphylococcal cross-infection will be serious.
 D. Aseptic Technic:
    1. Operating room - The operating room should be considered an isolation zone which may be entered only by persons wearing clean operating room attire (which may not be worn

elsewhere).

2. Ward procedures - All open wounds should be aseptically dressed to protect them from cross-infection and to prevent heavy contamination of the environment.

E. Nursing and Housekeeping:

1. Bedding must be laundered, and mattresses, furniture, and cubicles cleaned with a general utility antiseptic (see p. 125) after a patient is discharged.

2. Housekeeping procedures throughout the hospital must be thorough. Wet mopping and cleaning is required in order to prevent accumulation or raising of dust.

F. Antibiotics:

1. Prophylactic antibiotics should not be used.

2. Novobiocin, erythromycin, and chloramphenicol should be reserved for use in patients with staphylococcal infections except under unusual circumstances.

3. When possible, antibiotic therapy should be based on sensitivity studies.

4. Antibiotics should be given in adequate doses and discontinued as soon as possible.

G. Epidemiology:

1. Personnel with active staphylococcal infections should be excluded from patient contact until they have recovered. Personnel carrying staphylococci in their nasal passages or gastrointestinal tracts must observe personal hygiene, but need not be removed from duty unless they prove to be a focus of infection. The advisability of treatment of the carrier is uncertain since the carrier state is frequently transient or recurrent in spite of treatment.

2. Every significant staphylococcal infection acquired in the hospital should be investigated.

Treatment.

A. Local Treatment: Most staphylococcal infections (e.g., wound infections, furuncles) are superficial and are characterized by abscess formation. Treatment consists of rest, local heat, and drainage when localized.

B. Systemic Treatment: This is indicated when there are such signs of invasive sepsis as significant malaise and fever or an extensive local involvement. Under these circumstances antibiotic therapy is begun after adequate specimens for culture have been taken. If it is possible to wait, the antibiotics should be chosen on the basis of sensitivity studies. If delay is unwise, give (1) chloramphenicol, erythromycin, or novobiocin, and (2) one of the tetracyclines, subject to change upon receipt of sensitivity reports. Combined therapy with 2 or more drugs is advisable in serious staphylococcal infections.

ENZYMATIC DEBRIDEMENT

Proteolytic enzymes may be useful in the removal of surface exudates and necrotic tissue in decubitus, postphlebitic, varicose, and other indolent ulcers; and in wound infections, sinuses, abscess cavities, and third degree burns. When invasive infection is present, systemic antibiotics should also be used.

The enthusiasm for enzymatic debridement is by no means universal, but clinical experience with the following preparations shows that they effect more rapid removal of fibrin, pus, and eschar with earlier appearance of healthy granulation tissue.

A. Papain-urea-chlorophyllin ointment (Panafil®) contains papain as the principal proteolytic agent, with urea added to activate the enzyme and chlorophyll to reduce local irritation. The following prescription may be prepared by the pharmacy:

    ℞ Urea   10%
       Papain   10%
    In Chloresium Ointment®

    Sig.: Apply once or twice daily after irrigation or
            cleansing of the ulcer to remove surface debris

B. Ficin (bromelin) is a proteolytic enzyme from the fig tree available commercially in powder form as Debricin®, which may be mixed into a special jelly base or dissolved in normal saline. It is best applied thickly as the jelly to the ulcerated area, covered with vaseline gauze or a polyvinyl sheet, and the whole overlaid with a thick absorbent pad. In solution it may be instilled through catheters every 3-5 hours into a wet dressing covered with polyvinyl sheeting to limit evaporation.

C. Streptokinase and streptodornase (Varidase®) or purified crystalline trypsin (Tryptar®) may be sprinkled on the ulceration once or twice daily and covered with an occlusive saline dressing.

ANTIMICROBIAL THERAPY OF SURGICAL INFECTIONS*

Principles of Therapy.

A. Identify the infecting organism and choose an antimicrobial drug on the basis of sensitivity studies whenever possible. Once bacteriologic specimens are obtained it is justifiable, when necessary, to start therapy on the basis of clinical judgment, although the choice of drug may have to be reconsidered when laboratory investigation is complete.

B. Treat clinically significant infections only. Minor or superficial infections are best treated with conservative local measures. Indiscriminate use of antimicrobial agents may lead to toxic reactions, drug sensitization, the emergence of resistant

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*Anti-infective drugs and dosages are listed in the Appendix, pp. 614-621.

strains of bacteria, and disturbances in the mechanisms of host resistance.

C. Give adequate doses of an effective drug and discontinue administration as soon as possible. In most surgical infections, response will be apparent within 5 days if the drug and dosage are properly chosen and if there are no undrained foci of infection.

D. Use combinations of antimicrobial agents only when required (1) to treat mixed infections, (2) to prevent emergence of resistant mutants (as in antituberculosis therapy), or (3) to obtain additive or synergistic effects in overwhelming infections (e.g., staphylococcal meningitis). Combined therapy is particularly important in serious staphylococcal infections and in tuberculosis.

E. Do not substitute chemotherapy for definitive surgical measures such as the drainage of abscesses.

F. Avoid "prophylactic" antibacterial therapy to prevent infection in clean wounds.

G. Avoid topical administration of absorbable systemic drugs (especially penicillin and sulfonamides). Such "sensitizing" applications may form the basis of a severe hypersensitivity reaction upon later systemic use.

Drug Resistance.

An infectious organism may be naturally resistant to a given drug, may acquire resistance as a result of prior exposure, or may acquire resistance as a result of prior exposure to a similar drug (cross-resistance). Drug-resistant organisms arise either by mutation or by adaptation to the antimicrobial environment.

The emergence of resistant organisms can be reduced by the application of the following empiric rules.

A. Adequate Dosage: High tissue levels of drug inhibit both the original population and certain of the mutants.

B. Combined Therapy: If 2 drugs which do not give cross-resistance are administered simultaneously, each drug might delay the emergence of mutants resistant to the other.

C. Reserved Indications: Resistance can be minimized if 1 drug is reserved for use against a highly pathogenic organism or strain, e.g., novobiocin combined with other drugs for use in life-endangering hemolytic staphylococcal infection. Physicians should cooperate in attempts to reserve such drugs for specific purposes even though the drug may be quite effective or even superior when used against other more common but less dangerous pathogens.

Antibiotic Sensitivity Testing.

Sensitivity tests should be done whenever there is any doubt regarding the drug of choice.

A. Plate Test: This is a crude, rapid test performed by noting the zone of inhibition of bacterial growth around small paper disks saturated with various antibiotics and placed on the surface of the culture plate. This test does not always correlate well with the clinical response or with the results of tube sensitivity tests, but it is generally true that drugs failing to give

a zone of inhibition are not likely to be useful against the organism.

B. Tube Test: This test, which is performed in the test tube with standardized quantities of bacteria and antibiotic, more accurately estimates the antibiotic concentration required to inhibit or prevent growth of the organism. The tube test is the preferred method of sensitivity testing in serious infections where choice and dosage of antibiotic drug are critical factors in successful treatment.

7 . . .

Pediatric Surgery

Special Considerations in Pediatric Management.

Infants and small children have a relatively low tolerance to infection, trauma, blood loss, and nutritional and fluid disturbances. The management of these derangements is somewhat different in infants and children than in adults, and the margin of safety is narrower. Specialized knowledge and experience is required, and the collaboration of a pediatrician is valuable. Certain unique aspects of care in infants deserve emphasis.

A. Nutrition:
1. Preoperative feeding - Omit food for as brief a period as possible preoperatively to avoid hunger, restlessness, and glycogen depletion, which occur rapidly. An infant's last bottle, consisting of sweetened water or orange juice, should be given 4 hours before operation. Older children may have clear liquids until 2:00 a.m. preoperatively.
2. Routine vitamins - All infants should receive a daily vitamin supplement, orally if possible
3. Vitamin C is essential to wound healing. Give 25-100 mg. (3/8-1$^{1/2}$ gr.) orally or parenterally daily in addition to routine vitamins.
4. Vitamin K - Newborn infants frequently have a mild bleeding tendency caused by transient hypoprothrombinemia. Give Menadione Sodium Bisulfite, U.S.P. (Hykinone®), 5 mg. ($^{1/12}$ gr.) I.M. daily preoperatively and postoperatively.

B Parenteral Fluids: Quantity must be properly adjusted to the age and weight of the patient (see p. 99). **Maintenance of fluid and electrolyte equilibrium is of critical importance.**
1. Intravenous -
   a. Cutdown - Insert a small polyethylene catheter into an antecubital vein or the saphenous vein at the ankle or in the groin by cutdown technic preoperatively if blood or significant fluid replacement is expected. Avoid excessive fluid administration by careful monitoring of the intravenous drip. The cutdown catheter can be disconnected from the infusion and kept plugged for 6-12 hours at a time if filled with heparin solution (10 mg./ml.).
   b. Scalp vein - Peripheral veins are frequently inaccessible except by cutdown in small infants. A scalp vein may then be used. Venipuncture is made with a short-beveled 24 gauge needle. If gravity flow is inadequate, a three-way stopcock may be incorporated into the system for pumping fluid or blood.
2. Hypodermoclysis - 30-40 ml./Kg. body weight may be given in a single injection into the subcutaneous tissues of the axilla, upper back, thighs, or lower abdomen, using a 50 ml. syringe and 20 gauge needle. The addition of 150

Thyroglossal Duct Cyst
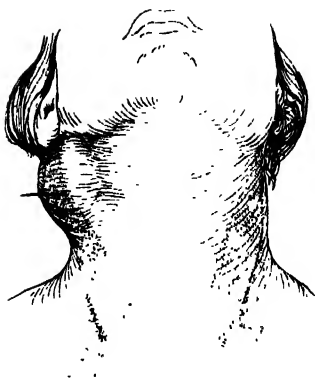
Branchiogenous Cyst
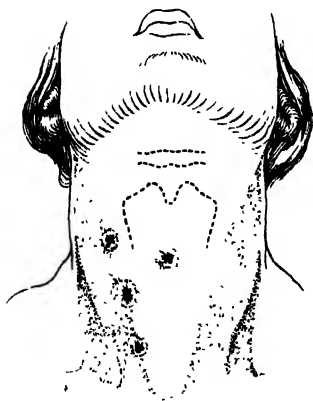
**Branchiogenous Fistulas
(Anterior View)**

**Branchiogenous Fistulas
(Median Sagittal View)**

turbidity-reducing units of hyaluronidase (Wydase®) to each 100-200 ml. of solution (e.g., physiologic saline or a mixture of equal parts of physiologic saline and 5% glucose in water) increases the rate of absorption Continuous hypodermoclysis can be given by a gravity apparatus with a Y-tube and 2 needles. Careful aseptic technic is essential in all of these procedures, and all solutions used must be isotonic.

C. Reduction of Fever: Children with high fever are poor operative risks since they are prone to develop marked tachycardia, convulsions, or collapse. Rectal temperature should be reduced to less than 102°F. (38.9°C.) preoperatively by sponging with tepid water or alcohol, fanning, rehydration, and antiinfective therapy as indicated Administer a salicylate as an antipyretic if necessary. Either of the following is adequate:

1 Sodium Salicylate Injection, N.F , 0.5-1 Gm. ($7^{1}/2$-15 gr.) I.V.

2 Acetylsalicylic acid (aspirin), orally or by rectum, and repeat for several doses p.r.n. Average dosage:

   0-2 years: 50-100 mg. ($^{3}/4$-$1^{1}/2$ gr.)
   2-5 years: 100-300 mg. ($1^{1}/2$-5 gr.)
   5-12 years· 300-600 mg (5-10 gr.)

D. Maintenance of Body Temperature: The temperature-regulating mechanism of small babies is inefficient. On the wards, in transit to the operating room, and in the operating room, severe falls in body temperature may occur. Take rectal temperature as frequently as required to detect and follow such changes. If possible, maintain operating room temperature at 68°F. (20°C.), and, when necessary to conserve the infant's body heat, wrap his legs and arms with cotton sheet-wadding. Warm blankets and a well-wrapped warm-water bottle may also be useful before and after surgery. An incubator should be used for premature infants.

DISORDERS OF THE HEAD AND NECK

BRANCHIOGENOUS CYSTS AND FISTULAS

During the first month of fetal life the primitive neck develops 4 external branchial clefts. Each cleft overlies an outpocketing of the foregut, the pharyngeal pouch, so that the external cleft is separated from the internal pouch by only a membrane. The ridges between the clefts are known as branchial arches, and these ultimately form portions of the face and neck. The first arch becomes the upper lip, cheek, maxilla, mandible, submaxillary gland, tragus, and helix. The second branchial arch forms the antitragus, antihelix, and lesser horn of the hyoid bone. The third arch gives rise to the greater horn of the hyoid bone and the skin of the mid neck.

Anomalies of the branchial apparatus include the following: (1) branchiogenous cysts (lateral cysts, congenital cysts of the neck, branchial cysts), with linings of stratified or columnar epithelium; (2) branchiogenous fistulas (lateral cervical fistulas, persistent branchial clefts, branchial fistulas), with linings of stratified squamous epithelium; and (3) preauricular and auricular sinuses.

Branchiogenous fistulas and cysts have a bilateral incidence of approximately 10%.

Clinical Findings.
- A. Cyst: Localized swelling anterior to the sternocleidomastoid muscle gives the impression of a solid mass because of the thick wall. There is no pain unless secondary infection has occurred, in which case erythema and tenderness are present. If a fistula opens into the pharynx, a sour taste may be noted if the cyst discharges into the mouth.
- B. Fistula: Mucoid material or crusting may be observed at a pinpoint skin opening over the middle or lower third of the sternocleidomastoid muscle. The central end of the fistula may communicate with the pharynx in the tonsillar area or may end blindly at any point. Milking of the tract from above downward may produce mucoid material at the orifice of the fistula. If the fistula becomes secondarily infected, symptoms of inflammation are present.
- C. Preauricular and Auricular Sinus: This consists of a short blind sinus in the skin of the preauricular area or on the helix.

Treatment.
Surgical excision through a transverse incision is the treatment of choice. If the tract is long it may be necessary to place a second or third transverse incision above the first for ease of dissection.

THYROGLOSSAL DUCT CYSTS AND SINUSES

Thyroglossal duct cysts (see p. 140) may develop from cell rests at any point along the migratory path of the thyroid gland. If the cyst suppurates and drains externally, a fistula will result. The cysts contain mucoid material and are lined by columnar epithelium.

Thyroglossal duct sinuses may exist with or without an associated cyst. They are blind sinuses passing for a variable distance upward toward the foramen cecum from a small cutaneous orifice.

Clinical Findings.
- A. Cysts: Thyroglossal duct cysts may be asymptomatic, may be large enough to cause symptoms by pressing on adjacent structures, or, if infected, may be tender and have a discharge. They may be found in the midline of the neck anywhere from the submental region to the suprasternal notch, but are most commonly located at the level of the hyoid bone. They vary in size from a barely palpable nodule to a mass 3-4 cm. in diameter. Motion of the cyst with protrusion of the tongue is characteristic.
- B. Sinuses: Thyroglossal duct sinuses open to the skin at any point between the hyoid bone and the suprasternal notch. A

deeply placed cord of dense tissue passing upward in the neck with attachment to the hyoid bone establishes the diagnosis.

Differential Diagnosis.
Sebaceous or dermoid cysts may also occur in the midline but are attached to overlying skin, whereas thyroglossal duct cysts are not. Ectopic thyroid tissue is clinically difficult to distinguish from a cyst. The distinction can be quickly made by radioactive iodine uptake studies.

Treatment.
For removal of either a cyst or sinus, the central portion of the hyoid bone and a block of tissue to the base of the tongue must be excised to give optimum results.
  A. Cysts: Smaller cysts (less than 0.5 cm. in diameter) may be observed until an increase in size is noted, in which case excision is advised. Larger cysts are apt to become infected and should be removed. Incision and drainage of an acutely inflamed cyst may be necessary. The inflammatory component should be allowed to subside prior to definitive removal of the cyst.
  B. Sinuses: All sinuses should be excised because of the danger of recurrent infection.

Prognosis.
With complete removal of the tract to the foramen cecum, the prognosis is excellent. Cysts will recur in 1-3% of patients, presumably because a branch or arm of a sinus was overlooked at the first operation.

CYSTIC HYGROMA OF THE NECK

Cystic hygroma is a benign subcutaneous tumor which probably originates as a developmental anomaly of lymphatic channels. It is most commonly seen in the posterior triangle of the neck, but may also involve the axilla, chest wall, or groin. The gross appearance is that of a translucent, thin-walled cystic structure containing clear fluid.

Clinical Findings.
Usually there are no symptoms. Difficulty in swallowing or breathing occurs rarely. The tumor varies in size from a few cm. in diameter to a massive structure which deforms the entire lateral neck, supraclavicular fossa, and shoulder. Characteristically it is spongy, with indefinite borders which are difficult to palpate. The overlying skin is normal to touch but the underlying fluid may give it a slightly blue or grayish tint. The tumor transilluminates readily.

X-ray examination of the cyst may delineate displacement of the pharynx or esophagus and may show extension into the mediastinum. Injection of radiopaque material outlines the extent of the cyst but may not show outpocketings.
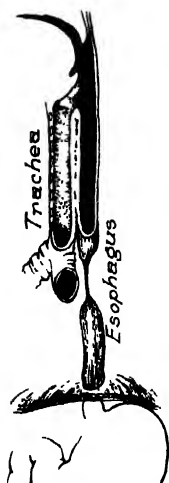
Esophageal Atresia
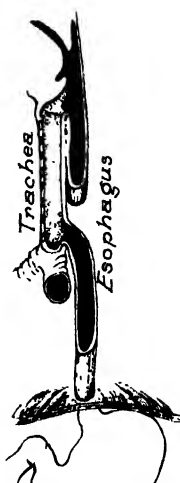
Tracheoesophageal Fistula With Blind Proximal Esophageal Pouch
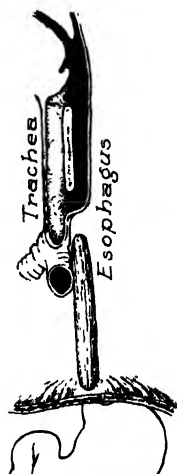
Tracheoesophageal Fistula With Blind Distal Esophageal Pouch

Tracheoesophageal Fistula With Proximal and Distal Esophageal Communication
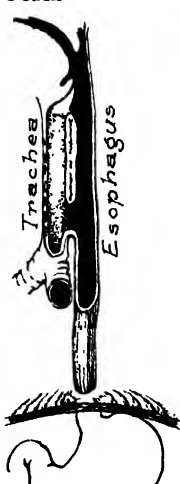
Tracheoesophageal Fistula With Intact Esophagus
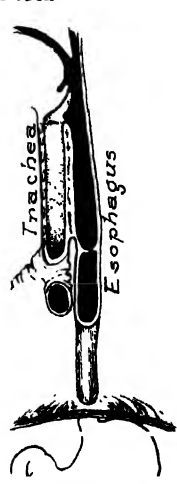
Esophageal Stenosis

Treatment.

Complete surgical excision is the treatment of choice, taking care to preserve vital neural and vascular structures. If excision is delayed, the tumor will only enlarge to the point where treatment is mandatory. Conservative measures have not been satisfactory: repeated aspiration carries the risk of infection; injection of sclerosing agents is dangerous because the cyst frequently is in contact with or surrounds the large vessels of the neck; and the tumor is generally conceded to be moderately resistant to irradiation, requiring high doses which hinder bone growth.

TONGUETIE AND RANULA

In tonguetie the frenulum linguae is abnormally short and the infant is unable to protrude his tongue. Treatment is not necessary unless there is difficulty in nursing. Speech is rarely impaired.

A ranula is a retention cyst of one of the sublingual salivary glands. The submaxillary glands are not involved, although the extensions of the ranula may lie in close proximity to Wharton's duct Ranulas vary in size with different degrees of ductal obstruction, and may occupy the whole floor of the mouth. Treatment is by surgical marsupialization or excision.

DISORDERS OF THE
ABDOMEN AND GASTROINTESTINAL TRACT

Examination of an infant suspected of having an acute abdomen is best carried out in a warm, quiet room Calm reassurance, pacifiers, and judicious fondling may facilitate examination. If possible, postpone examination of tender areas until the patient has relaxed, since small masses (e.g., the "tumor" of congenital hypertrophic pyloric stenosis) cannot be palpated if the abdominal musculature is tense.

CONGENITAL DISORDERS OF THE ESOPHAGUS

Normal embryologic development of the esophagus consists of progressive deepening of lateral furrows along a primitive tube of entoderm until the tube separates into the esophagus and trachea. Anomalous development results in atresia, fistula, or stenosis, or combinations of these defects (see p. 144).

The atretic esophagus is absent for a variable distance and may not communicate with the trachea. In the most common form (90%) of esophageal atresia, the distal segment communicates with the trachea. Less commonly, the proximal esophageal segment communicates with the trachea, or both segments are in tracheal communication. Rarely, a fistula connects the intact esophagus with the trachea. Stenotic narrowing is occasionally the only lesion.

Other anomalies which should be diagnosed prior to surgery are as follows (in order of decreasing frequency): congenital heart

disease, malformation of anus and rectum, Meckel's diverticulum, intestinal atresia or stenosis, malrotation of colon and intestines, coarctation of the aorta, annular pancreas, pyloric stenosis, harelip and cleft palate, and hypospadias.

Clinical Findings.

A. Symptoms and Signs: The child usually appears healthy and hungry. Saliva and mucus bubble from the nose or mouth. Feeding is promptly followed by gagging, coughing, regurgitation, and transient cyanosis as milk enters the bronchial tree.

A No. 10 or 12 F. urethral catheter passed through the mouth will fail to reach the stomach if the esophagus is atretic.

The degree of stenosis will determine how soon symptoms appear. Severe stenosis will cause vomiting and regurgitation soon after birth, whereas mild stenosis may be tolerated for years.

B. X-ray Findings: A small quantity (1-2 ml.) of iodized oil (Lipiodol®) or propyliodine (Dionosil®) placed in the esophagus via a catheter will demonstrate the upper blind pouch and, if a fistula is present, the tracheobronchial tree. A plain film of the abdomen gives valuable information about the lower esophageal segment: if no air is present in the stomach or intestines, there is no communication to the trachea; if air is present, an intact esophagus or communication with the trachea must be assumed.

Complications.

Pneumonitis secondary to spilling of saliva and milk into the tracheobronchial tree is common. Pneumonitis may develop late if feeding is delayed for 2-3 days.

Treatment.

A. Preoperative Care: Careful preoperative preparation is necessary even if surgery must be delayed 12-24 hours, as these babies may be debilitated from starvation and pneumonitis. The baby should be kept in a semi-reclining position to minimize the influx of gastric juice into the lungs. Constant suction on an indwelling catheter will keep the proximal esophageal segment empty. Anti-infective therapy (see p. 614) should be started as soon as the diagnosis is established. Parenteral nourishment (10% glucose in water, 1 ml./lb./hour) is imperative and should be administered via a cannula or a polyethylene catheter into an ankle vein. An oxygen tent may be indicated to relieve respiratory distress.

B. Surgical Treatment: Numerous multiple-stage surgical procedures have been devised to correct these anomalies, including closure of fistula, gastrostomy, and exteriorization of the proximal esophageal segment and the antethoracic esophagus. Current opinion holds that primary esophageal anastomosis within the chest is the best treatment since it achieves a one-stage union of the esophageal ends and obliterates the fistula. A Stamm feeding gastrostomy should be established at the end of the operation, since edema and temporary obstruction of the esophagus may occur during the healing phase.

C. Postoperative Care: Place the baby in an oxygen environment
with controlled humidity. Parenteral fluids and electrolytes
are given in accordance with physiologic and age requirements.
Excess saliva is removed from the mouth and nasopharynx by
suction whenever necessary. Feedings may be started through
the gastrostomy tube 48-72 hours after surgery, beginning with
10-15 ml. of 10% glucose in water at two-hour intervals and
advancing to formula on the fourth or fifth postoperative day.
When the child is swallowing saliva (i e., when suction is no
longer required), oral feedings may be started. This is usually
on about the eighth postoperative day. The feedings are in-
creased in quantity as tolerated and the gastrostomy tube is
then clamped. The tube is removed in 4 weeks. The opening
in the abdominal wall will close spontaneously. Specific anti-
biotics are continued for 5-7 days following surgery.

Prognosis.
   In experienced hands the mortality rate is about 30%. About
one-third of survivors will later require dilatation of a stricture at
the operative site.

ESOPHAGEAL STRICTURE

   Strictures of the esophagus may be congenital or acquired, and
can cause minimal to marked obstructive symptoms. Congenital
strictures are caused by failure of the esophageal anlage to form a
proper lumen. They may be formed by (1) a diaphragm or veil, with
an opening of varying size; or (2) a thickening of the esophageal wall
for a variable distance. Coexisting gastrointestinal anomalies
should be sought for. Acquired strictures may be secondary to
esophageal surgery or penetrating injuries, or may be caused by
ingestion of corrosive chemicals.

Clinical Findings.
 A. Symptoms and Signs:
   1. Congenital stricture - Regurgitation is the most prominent
      symptom. Regurgitation becomes more pronounced when
      the child is shifted from liquid to semi-solid food. The
      esophagus is capable of dilating, and may become large
      enough to accommodate a moderate quantity of liquid or
      semi-solid food without causing regurgitation. If dilatation
      is marked, the esophagus may press on the tracheobronchial
      tree and cause respiratory distress.   ,
   2. Acquired stricture -
      a. After esophageal surgery - The onset of vomiting or
         dysphagia in a child who has had esophageal surgery may
         be immediate (within 1-5 weeks) or delayed. The immed-
         iate type`is difficult to manage because symptoms are due
         to inadequate technic at the anastomosis (poor approxima-
         tion, leakage, etc.), which results in extensive fibrosis
         which does not readily yield to treatment. Delayed onset
         of symptoms is due to a fibrous ring in an otherwise
         essentially normal esophagus.
      b. After penetrating injuries - Excruciating pain and vomiting.

    c. After caustic burns of the esophagus - The patient who is
       seen shortly after swallowing corrosive material may be
       in shock. In the late phase, after the stricture is estab-
       lished, there will be dysphagia, inability to swallow
       (solids cause more difficulty than liquids), poor develop-
       ment, failure to gain weight, and mild anemia.
B. X-ray Findings: Contrast films, usually with iodized oil
   (Lipiodol®), will show the stricture and proximal dilatation.

Treatment.
A. Congenital Stricture: If a course of 8-12 dilatations fails to
   relieve symptoms, resection of the stricture with end-to-end
   anastomosis is indicated. A feeding gastrostomy should be
   considered when nutrition is poor and adequate intake cannot
   be maintained through the strictured esophagus. If relief is
   obtained from dilatations, these may be repeated indefinitely
   as often as symptoms require.
B. Acquired Stricture:
   1. After esophageal surgery - Immediate postoperative stric-
     tures are treated first by minimizing esophageal activity.
     This entails a liquid diet supplemented with parenteral or
     gastrostomy feedings. If symptoms persist after 4-6 weeks
     of such a regimen, esophageal dilatation is in order. This
     may be retrograde (through the gastrostomy) or from above,
     and is best done under anesthesia. The number and fre-
     quency of dilatations is determined by response.
   2. Resulting from penetrating injuries - The treatment is
     essentially the same as above.
   3. Resulting from caustic burns of the esophagus - Give anal-
     gesics for pain and small amounts (3-5 ml.) of a bland oil
     at hourly intervals during the first day to soothe the burns
     of the oral mucous membranes. Oral fluids are given as
     soon as the patient will accept them and a feeding gastros-
     tomy is performed in 5-7 days if fluids are not being swal-
     lowed.

     Passage of a tube for purposes of lavage carries the
     risk of esophageal perforation through a burned area.

     Esophageal dilatations are started about 4-6 weeks after
     the injury (some authorities favor dilatation as early as 2-3
     days following injury) and are continued as necessary there-
     after to keep the patient free of dysphagia. Retrograde dila-
     tation is preferred.

     Although repeated esophageal dilatation is a disagreeable
     prospect for the patient and thus makes a definitive surgical
     approach attractive, the results of esophageal replacement
     in children leave much to be desired. The procedures which
     have been suggested are (1) resection of the strictured
     esophagus with either intrathoracic or extrathoracic jejunal
     or colonic substitutes, and (2) high intrathoracic esophago-
     gastric anastomosis. In general, nonoperative treatment
     should be pursued to the point of maximum usefulness in an
     effort to avoid surgery.

HYPERTROPHIC PYLORIC STENOSIS

Hypertrophic pyloric stenosis is a congenital familial disorder of unknown etiology. It is characterized microscopically by an increase in the number and size of smooth muscle cells at the pylorus The symptoms are those of obstruction. It is more common in male infants (4·1)

Clinical Findings.
   A. Symptoms: The infant is normal at birth, but symptoms (regurgitation, progressing to projectile vomiting) develop within the first 8-10 weeks of life   Other symptoms (weight loss, constipation, dehydration, alkalosis, etc.) are secondary to the vomiting.
   B. Signs· Dehydration and wasting depend upon the severity and duration of the obstruction. The presence of a smooth, hard mass (like an olive) deep in the epigastrium to the right of the midline establishes the diagnosis. Peristaltic waves over the stomach may be visible. Absence of bile in the vomitus is a diagnostic aid.
   C. X-ray Findings: Barium study will show a dilated stomach and a long, narrowed pyloric canal.

Differential Diagnosis.
Hypertrophic pyloric stenosis must be differentiated from other causes of vomiting, e.g., intracranial hemorrhage or injury, stenosis of any portion of the gastrointestinal tract, malrotation, infection, and too-early formula advancement.

Treatment.
Correction of dehydration and electrolyte imbalance is important. Pyloromyotomy (Ramstedt) gives excellent results. This consists of longitudinal separation of the hypertrophied muscle fibers to increase the diameter of the pyloric canal   Feedings may be started within 4-6 hours after surgery   Begin with 30 ml. of 5% glucose in water every 2 hours for 4 feedings. Regular formula may then be substituted, beginning with small amounts every 2 hours and increasing until a normal quantity is taken.

Prognosis.
Pyloromyotomy mortality rates of less than 1% are common. Relief of symptoms is prompt and complete following operation.

OBSTRUCTION OF THE DUODENUM

Obstruction of the duodenum may be intrinsic, as a result of atresia or stenosis, or extrinsic, due to duplication, annular pancreas, or malrotation. About 20% of all gastrointestinal atresias and 50% of stenoses are in the duodenum.

Atresia and stenosis of the duodenum are caused by faulty embryologic development. Atresia is of 2 types: (1) a completely obstructing diaphragm or veil, and (2) discontinuity of the duodenum with a proximal blind pouch. Atresia is most commonly located distal to the ampulla of Vater. Stenosis may vary from a mild

DIFFERENTIAL DIAGNOSIS OF INTESTINAL OBSTRUCTION

	Age at Onset	Symptoms, Signs, and Laboratory Findings	X-ray Findings
Congenital diaphragmatic hernia	May be at any time, but usually neonatal.	Cyanosis, tachypnea, vomiting. Intestinal peristalsis in chest.	Abdominal viscera in chest.
Pyloric stenosis	4-10 weeks.	Projectile vomiting of gastric contents. Olive-like mass in area of pylorus.	Narrowing and elongation of pylorus.
Foreign body	Any age.	None, or vomiting and pain. No signs unless perforation occurs, then evidence of peritonitis.	Barium fluoroscopy may be necessary for nonopaque f.b.
Annular pancreas	Any age.	Vomiting; material may or may not be bile-stained, depending on location of pancreatic tissue. Abdominal distention (particularly epigastric).	Duodenal notching or constriction.
Duodenal atresia	First day of life.	Vomiting of bile-stained material. Epigastric distention. Absence of epithelial cells in meconium.	Distention of stomach and first part of duodenum.
Duodenal stenosis	Any age	Vomiting. If onset is immediately after birth, it cannot be clinically distinguished from duodenal atresia. Abdominal distention.	Duodenal distortion.
Malrotation	Any age.	Vomiting of bile-stained material. Shock may be present. Abdominal distention. If necrosis of intestine has occurred there may be rectal bleeding.	Stomach and duodenum may or may not be dilated.
Meconium ileus	First few days of life.	Vomiting of bile-stained material. Palpable loops of bowel	Granular appearance of air-meconium mixture.
Atresia of intestine and/or colon	First day of life.	Vomiting of bile-stained material. Abdominal distention. Absence of epithelial cells in meconium.	Massive distention of abdomen. No gas distal to atresia.
Congenital bands	Any age.	Vomiting, usually bile-stained. Abdominal distention. Rigidity and tenderness suggest perforation.	Dilated loops of bowel.
Intussusception	Most common between 6-12 months.	Intermittent abdominal pain followed by vomiting and occasional blood in the stool. Ovoid mass may be present.	Dilated loops of bowel may be present. Barium enema may show "meniscus sign."

asymptomatic narrowing to marked constriction with an opening only a few mm. in diameter.

Clinical Findings.
 A. Symptoms and Signs:
  1. Atresia - Vomiting of bile-stained material on the first day of life is the cardinal symptom. Vomiting becomes more frequent and more severe with subsequent feedings. Abdominal distention is limited to the epigastrium, and may not be present if the stomach is emptied by vomiting. Massive distention suggests perforation with peritonitis. Peristaltic waves may cross the epigastrium. Examination of the meconium for squamous epithelial cells will yield information of value. The epithelial cells are desquamated from the fetal skin and are a constituent of amniotic fluid. The meconium of a normal infant contains a great many of these cells, whereas in atresia the cells will not pass beyond the point of obstruction. Dehydration and alkalosis depend upon the duration and severity of vomiting.
  2. Stenosis - When symptoms appear in the neonatal period it is clinically difficult to distinguish stenosis from atresia. One month to several years later the symptoms (occasional vomiting, abdominal pain, and slow weight gain) may be so nebulous as to obscure the diagnosis.
     Signs are secondary to the degree of obstruction. The signs are more apt to be marked in the younger patient and less pronounced in the older patient.
 B. X-ray Findings: If the stomach has been emptied by vomiting, the x-ray may be essentially normal. Otherwise, gaseous distention of the stomach and proximal duodenum will be noted. In atresia there will be no gas in the intestine.
     A thin mixture of barium may be given if the diagnosis cannot be settled without contrast medium.

Treatment.
   Duodenojejunostomy, using a side-to-side anastomosis, is the surgical procedure of choice. Other areas of stenosis or atresia, particularly in the small intestine, should be carefully sought at surgery.

Prognosis.
   An appreciable mortality rate (25-35%) reflects the severity and multiple problems attending management of these patients . Prognosis for those who survive is good if there are no other anomalies.

MALROTATION (INCOMPLETE ROTATION)
OF THE INTESTINES

   As the fetus develops there is normally a disproportionate rate of growth between the alimentary tube and the celomic cavity. The faster-growing intestinal tract leaves the abdomen and enters the yolk stalk. When the abdominal cavity is large enough to accommodate the gut it returns from the yolk stalk in a definite sequence:

Hypertrophic Pyloric Stenosis
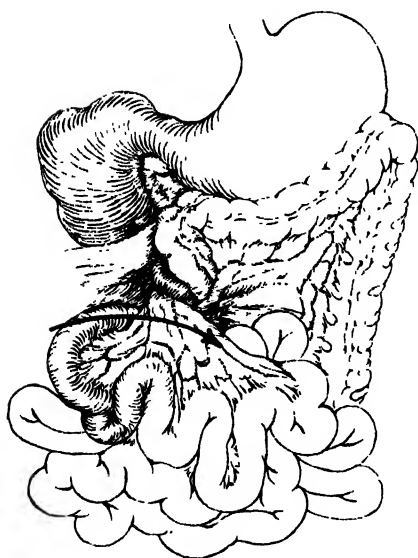
Malrotation With Midgut Volvulus, With Peritoneal Band Obstructing Duodenum. (Arrow shows direction of volvulus.)
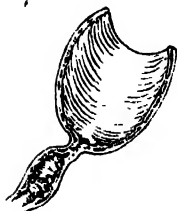
Intestinal Atresia

(1) The jejunum enters first, rotating counterclockwise until the distal duodenum is posterior to the mesenteric vessels. (2) The cecum follows and is carried to the right side of the abdominal cavity. (3) Rotation is complete when the mesentery of the right colon and small bowel are fixed in the adult position by attachment to the posterior wall of the abdominal cavity.

Failure to complete this process gives rise to incomplete rotation (malrotation). (1) In incomplete rotation the cecum fails to complete the final phase of rotation and comes to lie in the midline of the abdomen just below the stomach. Peritoneal attachments run from the cecum to the right posterolateral abdominal wall and pass over the distal portion of the duodenum, resulting in duodenal obstruction. (2) In volvulus of the midgut the mesentery of the small bowel has only a rudimentary superior attachment to the posterior wall of the abdominal cavity. The entire length of the small intestine swings from this short attachment and can easily twist on itself. A volvulus is established when the small intestine winds about itself and the cecum. (3) In mobile cecum the mesentery of the cecum and terminal ileum fail to attach, resulting in a normally placed but abnormally mobile cecum and terminal ileum.

The form of malrotation most commonly seen in children is that causing duodenal obstruction, and in this group about one-half will have an associated midgut volvulus.

Clinical Findings.

A. Symptoms and Signs: 80% of children with malrotation will present with symptoms during the first year of life which require surgery. Vomiting of bile-stained material, development of epigastric fullness and tympany, and visible gastric peristalsis suggest duodenal obstruction. Dehydration, alkalosis, and malnutrition are secondary to vomiting. If necrosis of the gut has occurred, there will be rectal bleeding.

Symptoms in the older child are characterized by intermittency over a long period of time. Abdominal pain and nausea and vomiting may not be severe enough to cause the parents to seek medical attention. On the other hand there may be a crisis requiring surgery.

B. X-ray Findings: Findings vary with the degree of duodenal and intestinal obstruction. If vomiting has emptied the stomach, nothing significant will show on the film. However, the stomach and duodenum may be massively dilated. Small amounts of dilute barium by mouth may show a duodenal obstruction or loops of small bowel anomalously placed on the right side of the abdomen. A barium enema which shows a medially placed appendix and cecum aids in establishing the diagnosis.

Treatment.

A. Preoperative Care: Preoperative care consists of gastric deflation by means of a nasogastric tube and correction of dehydration with intravenous fluids.

B. Surgical Treatment: At surgery the midgut volvulus will be obvious; however, the possibility of incomplete rotation of the cecum must be investigated and corrected if present. After the volvulus has been untwisted, the peritoneal bands which obstruct the duodenum must be transected so as to allow the cecum and

terminal ileum to lie on the left side of the abdominal cavity.

If surgery is undertaken for signs and symptoms of mal-
rotation and an abnormally mobile cecum is encountered, the
mesenteric attachment to the cecum determines the surgical
procedure. If the posterior attachment of the mesentery is
relatively long, the cecum may be sutured to the posterior ab-
dominal wall. If the attachment is short, the leaves of the
mesentery should be divided to allow the cecum and right colon
to fall to the left side of the abdominal cavity.

ATRESIA AND STENOSIS OF THE
INTESTINE AND COLON*

Atresia is due to failure of the embryologic process to form a
lumen. Stenosis consists of incomplete canalization and consequent
narrowing of the lumen. The atretic bowel may have an obstruct-
ing diaphragm or veil, or may exhibit discontinuity with a proximal
blind pouch. About 50% of all gastrointestinal atresias and stenoses
will be in the ileum.

The clinical picture in the newborn period is that of complete
intestinal obstruction. Vomiting and abdominal distention are the
prominent symptoms. If the infant is a few weeks old the present-
ing symptoms of stenosis are less marked, and there may be a
history of several formula changes in an attempt to control the in-
termittent vomiting. In older children (1-12 years) the complaints
are vague: intermittent abdominal pain, occasional vomiting, and
poor weight gain.

Plain films of the abdomen show marked dilatation of the duo-
denum and distention of the proximal intestine. There will be no
gas distal to the atresia.

An anastomotic procedure is the treatment of choice, but if the
bowel is so small as to make an end-to-end anastomosis dangerous,
or if the infant is critically ill, a side-to-side anastomosis is pre-
ferred. Occasionally a Mikulicz exteriorization may be necessary.

Mortality rates of approximately 25% are usual. If surgery is
successful, the outlook is excellent.

FOREIGN BODIES
OF THE GASTROINTESTINAL TRACT

If a foreign body reaches the stomach it will in all probability
pass through the rest of the intestinal tract; even open safety pins
commonly pass through. The mere presence of a foreign body is
therefore not a surgical emergency.

Serial x-rays (several times a week) should be taken to ob-
serve the progress of the foreign body. (Lead shielding of the
gonads is advisable.) Surgical removal is indicated when the for-
eign body has been stationary for several days or if clinical signs
of obstruction or perforation develop. However, foreign bodies
which lodge in the esophagus must be promptly removed by esopha-

*Duodenum, see p. 149.

goscopy since vomiting with aspiration or esophageal perforation with subsequent mediastinitis or pneumothorax can occur.

CONGENITAL BANDS

Obstructing bands (remnants of embryologic processes) in various locations may cause vomiting and abdominal distention and yield x-ray signs of intestinal obstruction. Treatment is by surgical division of the bands.

INTUSSUSCEPTION

Intussusception, usually seen in the first year of life, is invariably fatal if not reduced. Definitive therapy should be given as soon as the diagnosis is established. The defect consists of invagination or telescoping of proximal (intussusceptum) into distal (intussuscipiens) intestine or colon. Intussusception commonly occurs at the ileocecal junction. In approximately 95% of cases there is no agreement as to cause ("idiopathic"), although change of diet to solids, hyperperistalsis from any source, and gastroenteritis have been implicated. Recognized causes are Meckel's diverticulum, polyp, or tumor.

Clinical Findings.
  A. Symptoms: Characteristically there is a sudden onset of paroxysmal abdominal pain in a previously healthy child. During paroxysms the child will double over or draw up his legs, become pale, cry, and have grunting respirations. In the pain-free intervals the child will relax and appear healthy. Vomiting frequently ensues and gradually increases in severity. After 8-16 hours, blood is passed per rectum. The degree to which the blood supply of the intestine has been compromised determines the amount of blood passed; blood loss may be insignificant or may be sufficient to cause shock.
  B. Signs: The duration of symptoms determines the severity of shock, dehydration, tachycardia, and listlessness. A fusiform, elongated, or ovoid mass which is firm and nontender can often be palpated, usually in the right upper quadrant. A mass in the subhepatic space may not be detected, but in this instance the right lower quadrant is "empty" (Dance's sign).
  C. X-ray Findings: If there is a history of paroxysmal pain, progressive illness, and a palpable abdominal mass, x-ray examination is not necessary. If the diagnosis is in doubt, x-ray examination of the abdomen will yield signs of small bowel obstruction and a barium enema will outline a capping or meniscoid filling defect which obstructs the retrograde flow of barium.

Treatment and Prognosis.
  A. Conservative Measures: Interest in hydrostatic reduction, proposed in 1938 by Hipsley and more recently by Hellmer and Ravitch, has given rise to controversy regarding the merits of this approach as compared to surgery. Under fluoroscopic control an attempt is made to reduce the intussusception with

barium. Exploratory laparotomy has been necessary in 15-25% of the patients so treated to ascertain completeness of reduction. Mortality varies up to 5%.

B. Surgical Treatment: Reduction at surgery is favored by others (Gross, Scott, Swenson). Simple reduction is achieved by gentle compression of the intussuscipiens to slowly eject the intussusceptum. If the intussusception is irreducible or the bowel gangrenous, a resection is indicated. An end-to-end anastomosis is preferable, but exteriorization (Mikulicz) may be necessary if the patient is critically ill. The ileocolostomy or ileoileostomy can be closed in 7-10 days. Incidental appendectomy is contraindicated (may predispose to postsurgical infection) unless the appendix is gangrenous.

Reports from experienced surgeons show that mortality varies with the duration of symptoms. Surgery within 24 hours of onset has a very low mortality (Gross reports no fatalities). If treatment is delayed, the mortality increases. A 1-2% recurrence rate of intussusception may be expected.

MECONIUM ILEUS

Intestinal obstruction in the newborn secondary to inspissated meconium is an early manifestation of fibrocystic disease (mucoviscidosis). The essential defect is of the mucus glands of the gastrointestinal and respiratory tracts, which produce a scanty, viscid secretion. In particular, the pancreatic fluid is diminished in volume and deficient in enzyme content (trypsin). This results in a thick, tenacious, glue-like meconium which mechanically obstructs the intestine.

Clinical Findings.

A. Symptoms and Signs: Vomiting of bile-stained material occurs in the first 48 hours of life. The abdomen is distended and has a doughy texture. Loops of bowel are visible under the abdominal wall, and firm masses can be palpated within the intestinal loops. The infant usually fails to pass meconium.

B. X-ray Findings: Extensive dilatation of the intestines in association with a granular or mottled appearance of the dilated loops is characteristic of meconium ileus. The unique appearance is due to gas bubbles mixed with meconium.

Treatment.

If symptoms are mild, intermittent gastric suction with administration of pancreatic enzymes (Viokase®) by enema and gastric tube may relieve the obstruction. However, the vast majority of patients will come to surgery. The operations which have been tried include (1) ileotomy with removal of the obstructing meconium, with or without instillation of solutions to loosen the meconium; (2) exteriorization (Mikulicz); and (3) resection with anastomosis. Each of these procedures has its own limitations, and a rational approach is to progress from the simpler to the more complex procedure. For example, hydrogen peroxide can be instilled into the bowel by needle puncture in several areas in an attempt to distend the bowel and separate the meconium from the bowel wall. The meconium is

then evacuated from the bowel via multiple enterotomies. Should this fail, or if there is associated gangrene, perforation, or volvulus, a resection is mandatory. The simplest and safest method is exteriorization of the involved bowel, closure of the abdomen, and resection of the intestinal loops. The resulting ileoileostomy or ileocolostomy is closed in 7-10 days.

Postoperative treatment of mucoviscidosis consists of a low-fat, high-caloric, high-protein diet with water-miscible vitamins, pancreatic extracts, and antibiotics for pulmonary infections.

Prognosis.
Mortality in the best of hands is about 50%. Survivors must be observed carefully so that other aspects of fibrocystic disease can be treated.

MEGACOLON
(Hirschsprung's Disease)

Megacolon is caused by congenital absence of ganglion cells in Auerbach's plexus of the rectum or rectosigmoid, resulting in dysfunction of peristalsis in the aganglionic segment. Inability of coordinated peristalsis to pass through the aganglionic area causes the equivalent of a chronic partial obstruction. Fecal material accumulates and there is dilatation and hypertrophy of the proximal colon and abdominal distention. Males are more frequently affected than females (9:1). Although constipation is the rule, diarrhea may be present when liquid stool passes around the fecal mass in the colon.

The clinical picture depends upon the severity of the disorder. Occasional constipation and minimal abdominal distention are present in the milder forms. In the severe forms of the disease, profound fecal stasis can cause upward displacement of the diaphragm with atelectasis of the lung bases and pulmonary embarrassment which, when coexisting with cachexia and toxemia, may result in a critical situation necessitating immediate therapy.

Barium enema shows the aganglionic and distal segments to be of normal caliber, whereas the proximal portion of the colon is dilated and redundant.

If the diagnosis of megacolon is in doubt, rectal biopsy under general anesthesia is indicated to establish the presence or absence of ganglion cells.

Enterocolitis and ulceration of the mucosa may occur as a consequence of pressure necrosis from the hardened feces.

Treatment and Prognosis.
  A. Medical Measures: The mild forms of megacolon can be managed by diet supervision to avoid high-residue foods and by agents to soften and lubricate the stool. Frequent enemas are necessary. Parasympathomimetic drugs (Doryl®, Mecholyl Bromide®) are useful on occasion.
  B. Preoperative Care: The colon must be completely emptied and the gastrointestinal tract sterilized.
  C. Surgical Treatment: Cecostomy or colostomy is not definitive, but is a useful preliminary step until definitive surgery is

**Membranous
Imperforate Anus**
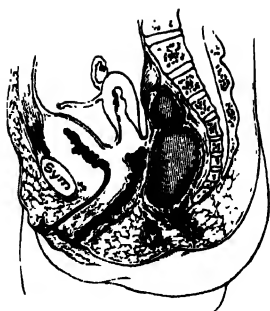
**Imperforate Anus With Rectal
Pouch Ending High Above Anus**
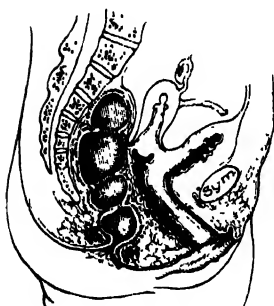
**Imperforate Anus
With Normal Anal Canal**

Rectovaginal Fistula
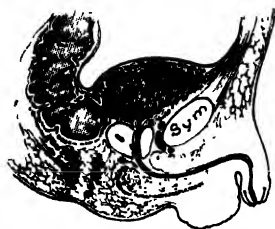
Rectovesical Fistula
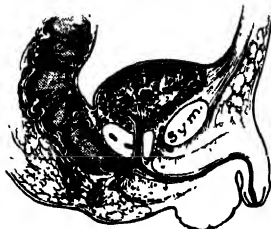
Rectourethral Fistula

technically feasible or as a life-saving procedure in a critically ill child.

The surgical procedure of choice is abdominoperineal removal of the rectosigmoid, the so-called "pull-through" operation (Swenson). By a combined abdominal and perineal approach, the dilated sigmoid is resected, the rectum and rectosigmoid everted through the anus and transected with anastomosis of the rectum and proximal colon 1-2 cm. above the anus.

Abdominoperineal resection and anastomosis will yield excellent results in 80% of cases.

ANORECTAL MALFORMATIONS

In the embryo, the urogenital and intestinal tracts have a common terminal cavity, the cloaca. The cloaca is normally separated by the downgrowth of a membrane which divides the urogenital system from the intestinal tract. A dimpling of the skin, the proctodeum, invaginates until it joins the rectum and the anal membrane ruptures to establish the anal canal. Arrested or faulty development may lead to anal stenosis or imperforate anus. (In anal stenosis the anal membrane is partially remnant.)

Imperforate anus may be of the membranous variety with a persistent anal membrane, may have a blind rectal pouch ending above the anus, or may show a normal anus and ampulla with a blind rectal pouch terminating in the hollow of the sacrum.

About 70% of patients with imperforate anus have fistulas from the rectum to the genitourinary system or to the perineum. Rectourethral and rectovesical fistulas are seen almost exclusively in male infants. Sex distribution of rectoperineal fistulas is equal.

Anorectal malformations occur once in 5000-7000 births.

The incidence of other congenital anomalies may be as high as 40%. The presence of associated anomalies should be documented, as their presence influences the outcome.

Clinical Findings.

The pattern of anomalies determines the symptomatology and the age at which the defect is recognized.

A. Symptoms and Signs: The symptoms are absence of bowel movements followed by abdominal distention and vomiting. Imperforate anus is readily recognized on examination. An imperforate anal membrane is dark because of the meconium lying against it. In the patient who has a rectal pouch ending several cm. from the skin, an anal dimple is often present. The dimple may vary from a true dimple to a depression that will admit the tip of a thermometer. A valuable aid in assessing the ultimate outcome is to note whether the skin surrounding the anal dimple puckers with the stimulation of examination or stroking. Puckering of the skin indicates contraction of the anal sphincter muscle; if this sign is present, the prognosis for control of defecation following surgery is good.

When the anal ampulla is normal and the rectal pouch terminates in the hollow of the sacrum, the cause of distention and vomiting may not be determined for several days.

When a fistula exists, meconium, stool, or gas (or all 3)

may be passed via the penis or vagina or from the cutaneous orifice of the fistula. A probe passed into the fistula while holding a finger on the anal skin will measure the distance between the anal skin and the rectal pouch. If a fistula to the skin is large, life can be supported for years.

In anal stenoses the symptoms are obstipation, stool of decreased size which may be flattened or "ribbon-like," and abdominal distention. On examination the orifice may measure only a few mm. in diameter.

B. Laboratory Findings: On occasion a small fistula will become plugged with meconium and cause no symptoms; in all patients with imperforate anus, repeated examinations of the urine sediment should be made to exclude this possibility. When a fistula is present, meconium detritus or fecal debris will ultimately be demonstrated in the urine.

C. X-ray Findings: X-ray examination is imperative because the distance between the anal skin and rectal pouch determines the surgical approach (abdominal vs. perineal). Holding the infant with his head down allows gas to pass upward into the rectal pouch; anteroposterior and lateral films will then accurately show the position of the rectal pouch. A lead marker placed on the anal skin or a thermometer inserted into the anal pouch will serve as a reference point for measurements. (Bear in mind that it may take 18-24 hours after birth for gas to pass through the meconium and reach the rectum.) Visualization of the fistula tract by the injection of radiopaque material is tedious and frequently gives imperfect results.

Treatment.

A thin, imperforate anal membrane can be treated by cruciate incision or by excision of any constricting membrane.

If the rectal pouch is within 1.5 cm. of the anal skin, a perineal approach can be undertaken to dissect the rectum, transect any fistulas, and suture rectal mucosa to skin. An interval of more than 1.5 cm. necessitates an abdominoperineal approach with dissection of the rectum and distal sigmoid, transection of a fistula, if present, and anastomosis of rectal (or distal sigmoid) mucosa to anal skin. This is a major procedure which should be undertaken only if the patient is free of associated anomalies and is in good condition.

Sigmoidostomy is the procedure of choice in a critically ill infant. The definitive procedure may be delayed 1-2 years until the growth pattern and health of the infant have been clearly established.

Anal stenosis is treated by repeated dilatations for as long as necessary.

Prognosis.

The surgical mortality in experienced hands is 5-10% if other complications are not present. A satisfactorily functioning anus is present after surgery in 90% of patients. The recurrence rate (due to infection) for fistulas is about 3%.

UMBILICAL HERNIA

Failure of closure of the fascial ring at the umbilicus causes a protrusion of varying size. It is commonly seen in Negroes, and more often in females than males (2:1).

There are usually no symptoms. The projection at the umbilicus is easily reducible and reappears upon the slightest effort. Incarceration of the intestines is a rare complication.

Because spontaneous closure occurs in a high percentage of cases, these patients should be observed periodically without treatment. Some authorities advise keeping the hernia reduced by taping a block of sponge rubber over the navel or infolding skin. Others doubt that strapping is of value.

If the ring increases in size or remains stationary without evidence of closing for 12-18 months, surgery is indicated. The preferred technic preserves the umbilicus and approximates the fascial ring in a vertical closure so as to allow any defect between the recti to be corrected.

OMPHALOCELE
(Hernia Into the Umbilical Cord, Umbilical Eventration)

During embryonic life a portion of the intestinal tract herniates into the umbilical cord, presumably because these viscera and the abdominal cavity have disproportionate growth rates. Further growth brings about relative enlargement of the abdominal cavity, and the intestines return to it. An omphalocele results when the umbilical orifice fails to close. The intestines as well as other abdominal organs may be displaced into the persistent sac. A thin membrane, lined internally by peritoneum and externally by amniotic membrane, comprises the omphalocele sac.

Clinical Findings.
An omphalocele sac containing abdominal viscera is usually the only finding unless there is a coexisting anomaly such as malrotation (may cause vomiting), congenital heart disease, and imperforate anus. Malrotation is present in about one-fourth of cases.

Complications.
The avascular nature of the thin omphalocele sac makes it vulnerable to bacterial invasion, and infection readily occurs if surgery is delayed.

Treatment.
Immediate surgery is indicated. Whether this should be done as a one-step or two-step surgical procedure depends upon the size of the omphalocele. In the smaller omphaloceles (less than 7 cm. in diameter), excision of the sac with layer closure of the abdominal wall is advised. In the larger omphaloceles (greater than 8-9 cm. in diameter), a two-stage procedure is necessary. This consists of a circumferential incision at the junction of skin and sac to allow undermining of the skin so that it may be closed over the intact sac. The second stage is carried out whenever the abdominal cavity readily accommodates all of the viscera. This may be within

12-24 months but may have to be delayed for as long as 2 years.
At the second operation, the peritoneal-cutaneous sac is opened
and excised, and a layer closure is carried out.

Prognosis.
Even in experienced hands the larger omphaloceles carry a
mortality rate of about 85% (Gross). Smaller omphaloceles have
a mortality rate of about 25%.

CONGENITAL ATRESIA OR STENOSIS
OF THE BILE DUCTS

The biliary system may be atretic or stenotic at any level as
a result of faulty embryologic development. Atresia is more com-
mon. About 15-20% of infants with atresia have surgically correc-
tible lesions. The remainder will have a pattern of intrahepatic and
extrahepatic atresia which cannot be corrected by surgery. About
one-third of extrahepatic atresias will show complete atresia of the
gallbladder and extrahepatic ducts; about one-third will have atresia
of the extrahepatic duct system with a patent gallbladder; and the
rest will be evenly distributed between atresia of the upper and
lower extrahepatic ducts, with a patent gallbladder connecting with
the normal portion of the duct system. The liver shows obstructive
biliary cirrhosis, and the spleen may be enlarged as a result of the
portal obstruction.

Clinical Findings
A. Symptoms and Signs: The cardinal symptom is jaundice, which
   is usually present at birth but which may not be apparent for
   1-4 weeks. Once established, the jaundice becomes progres-
   sively more intense and the skin ultimately takes on a deep
   yellowish-green tint. The stools are white, the urine is dark,
   and there may be a hemorrhagic phenomenon due to deficiency
   of vitamin K. Development is below normal in children older
   than 4-6 months. Physical and mental lethargy is notable.
B. Laboratory Examination: Blood clotting and prothrombin times
   are prolonged. Urinary bile pigments are present in large
   amounts. Urine urobilinogen is absent. The mucosa of the
   gastrointestinal tract may excrete small amounts of biliary
   pigments when there is intense hyperbilirubinemia, and the
   stool may therefore contain small amounts of bile pigments.

Differential Diagnosis.
Biliary obstruction may be confused with physiologic jaundice,
erythroblastosis fetalis, inspissated bile, choledochus cysts, and
hepatitis.

Treatment.
During the period when the diagnosis is in doubt, it is advisable
to attempt stimulation of bile flow by means of cholagogues (Patter-
son). Dehydrocholic acid (Decholin®) is administered intravenously
3 times a week, and Ketochol® is given with each meal. If a flow of
bile has not been produced within 3 weeks, surgical exploration is
advisable.

The pattern of atresia determines which types of operative pro-
cedures are indicated. When atresia is present, the recommended
procedures are hepaticoduodenostomy, cholecystoduodenostomy,
and choledochoduodenostomy. If the obstruction has been caused
by inspissated bile, the biliary tract should be flushed out with
normal saline by means of a catheter inserted into the gallbladder.
Operative cholangiograms give helpful information about the ductal
pattern.

Prognosis.
When the biliary obstruction cannot be relieved by surgery, the
disease is characterized by gradual deterioration and death by the
second or third year. In those patients with correctible lesions,
there will be an immediate mortality of about 35% and a delayed
mortality (due to liver damage) of about 25%. Those who survive
surgery and do not show evidence of liver impairment have an ex-
cellent prognosis.

CHOLEDOCHUS CYST

Choledochus cyst is a rare disorder of uncertain etiology char-
acterized by massive cyst-like dilatation of the common duct, which
may contain 1-2 L. of bile. The wall of the duct becomes thickened
and leathery. There may also be slight enlargement of the cystic
duct, hepatic duct, and gallbladder. The pathognomonic triad is
jaundice, abdominal pain, and a palpable tumor. Choledochus cysts
are more common in females (4:1).
Plain films of the abdomen show a soft tissue mass in the right
upper quadrant and a nonfunctioning gallbladder.
Treatment consists of anastomosis of the cyst to the duodenum
or small bowel   A mortality rate of 15-20% may be expected. For
patients surviving surgery the prognosis is excellent.

DISORDERS OF THE
EXTREMITIES

SUPERNUMERARY DIGITS

Supernumerary toes or fingers may be completely formed, with
joint surface and bone, or may consist of soft tissue only. Func-
tional impairment and a wider than normal foot (making shoe fitting
difficult) justify surgical removal.   •

WEBBED DIGITS

Congenital fusion of fingers and toes, resulting in a web be-
tween adjacent digits, is functionally and cosmetically undesirable.
Webs of the toes are usually ignored, as they cause no impairment;
surgical treatment is reserved for the occasional patient showing
a complication of the webbing process. Surgical treatment of webbed
fingers consists of separation of the fingers and construction of an
interdigital flap to give maximum mobility.

8 . . .

Surgery of the Head and Neck*

TRAUMA TO THE HEAD AND NECK †

EPISTAXIS

The most frequent cause of epistaxis at any age is spontaneous erosion of 1 of the superficial mucosal blood vessels situated over the cartilaginous nasal septum (Kiesselbach's area) or the anterior end of the inferior turbinate. External trauma to the nose with or without fracture is a frequent cause. Minor trauma such as nose-picking may lead to ulcerations of the nasal septum and subsequent hemorrhage Less common causes are neoplasms of the nasal chamber or paranasal sinuses, and spontaneous rupture of a branch of the ethmoidal or sphenopalatine arteries or of vessels within the meatuses.

Acute infectious diseases of childhood, hypertension, arteriosclerosis, cardiac disease, and blood dyscrasias and coagulation defects may be associated with epistaxis.

Treatment.
A. Specific Measures: Treat the underlying disease; give transfusions as necessary if blood loss is excessive.
B. Local Measures: Have the patient sit up and forward to prevent swallowing and aspiration of blood.
  1. Anterior epistaxis -
    a. Pressure over the area (pinching the nose) for 5 minutes is usually sufficient to stop bleeding. This may be combined with packing the bleeding nostril with cotton moistened with saline solution or 1:1000 epinephrine solution.
    b. After active bleeding has ceased (or if pressure fails to stop the bleeding), a cotton pledget soaked with 2% tetracaine (Pontocaine®) is applied to the bleeding area and the ruptured vessel is cauterized with a chromic acid bead or an electrocautery needle. Chromic acid must be neutralized immediately with saline solution. After 24 hours, zinc oxide ointment is applied to relieve crusting. Repeat cauterization is infrequently necessary.
    c. If the source of bleeding is not accessible to cauterization (beneath the middle or inferior turbinates, behind septal spurs, or high in the vault), the nasal chambers must

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*Congenital anomalies of the head and neck are discussed in Chapter 7, Pediatric Surgery.
†The principles of treatment of soft tissue injuries are discussed in Chapter 1, Trauma and Emergencies. Other fractures are discussed in Chapter 19, Traumatic Orthopedic Surgery.

be anesthetized topically and packed with half-inch selvedged gauze soaked in cod liver oil. The packing is placed in laminated layers, starting either in the vault or on the floor of the nasal chamber. Anchor the pack by means of a string taped to the face. It is safe to leave the nasal packing in place for as long as 5 days provided the patient is given adequate antibiotic medication to help prevent suppurative otitis media and sinusitis.

2. Posterior (nasal pharynx) epistaxis - In posterior epistaxis it may be necessary to introduce a posterior nasal pack. This is done by sewing 3 strings (braided 0 silk) through and through the center of a rolled 4 × 4 gauze sponge. A soft rubber catheter is passed through the nostril and out through the mouth. Two strings are attached to the catheter and drawn out through the bleeding nasal chamber. The pack is guided by the finger into the nasal choana posteriorly. The 2 strings are anchored over a gauze bolster at the nares. The third string is allowed to remain in the mouth and is anchored to the face, to be used later for removing the pack. The pack should not be left in place more than 3 days. Examine the patient's ears daily for evidence of otitis media. Hemorrhage may recur when the packing is removed or may even continue with the packing in place. If this happens, the pack must usually be changed or reinserted under general anesthesia.

3. If the bleeding persists from a source low in the nasal chamber (root of the inferior turbinate or below), external carotid artery ligation in the neck must be considered. Uncontrolled bleeding from high in the vault of the nose may have to be controlled by ligation of the anterior or posterior ethmoidal artery (or both) as it passes from the orbit into the ethmoidal labyrinth.

FRACTURE OF THE LARYNX

Fracture of the larynx is caused by direct blunt trauma to the anterior part of the neck. The thyroid cartilages are usually fractured. In more severe injuries, the cricoid cartilage as well as the cartilaginous tracheal rings are crushed. The arytenoid cartilages may be displaced. There are symptoms and signs of laryngeal obstruction. Emphysema of the neck tissues is usually evident.

Absolute signs of laryngeal fracture are lacerations of the mucosa of the larynx and blood expectoration as noted by either direct or indirect examination. •

X-rays of the neck may show gross dislocation of laryngeal structures, narrowing of the airway, and edema and emphysema of the soft tissues. The diagnosis of laryngeal fracture should not depend on x-rays and should be made clinically.

Emergency care is life-saving. **Establish an airway** (usually by tracheostomy; see p. 166).

Open reduction is the treatment of choice of laryngeal fractures. After the fragments have been reduced, a splint (polyethylene tube or acrylic dilator) should be placed in the larynx and anchored through the neck tissue with wire or to the tracheostomy cannula
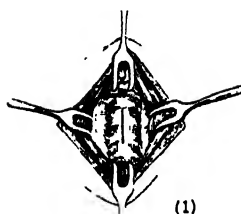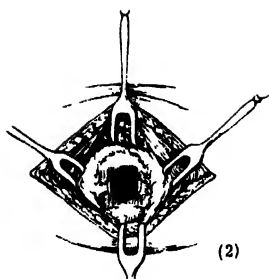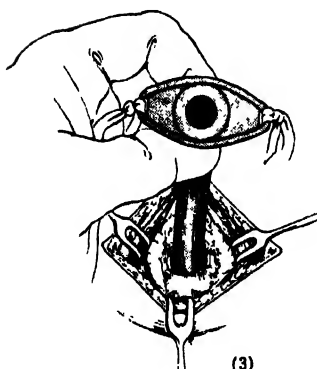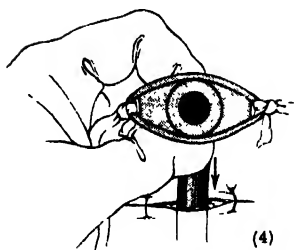
Technic of Tracheostomy.
  (1) Surgical approach in children. Third and fourth tracheal rings split in midline. No cartilage removed.
  (2) Surgical approach in adults. Anterior section of third tracheal ring removed.
  (3) Insertion of tracheostomy tube.
  (4) Loose suturing of both ends of wound.
  (5) Tube tied in place.

with braided silk ligatures. In most cases the splint must be worn for 3-6 months before it is removed. **Caution:** Late reduction due to failure to recognize laryngeal fractures may lead to serious stenosis of the larynx.

INFECTIONS OF THE NECK*

CHRONIC INFLAMMATION OF THE SALIVARY GLANDS
(Chronic Parotitis, Sialadenitis, Sialectasia)

Chronic inflammation of the parotid gland may be secondary to single or multiple calculi or strictures of the duct system, or may occur without apparent cause. There is a recurrent swelling of the gland for months or years which is more pronounced after meals. Chronic enlargement and induration of the gland develops until finally the interlobular ducts become enlarged and sacculated, with lymphoid infiltration of the lobules. Thick, tenacious, or purulent secretions may be expressed from Stenson's duct.

X-rays may show calculi. Sialograms often show single or multiple strictures and sacculations of the interlobular duct system.

Dilatations of Stenson's duct, intraoral removal of stones, and meatotomy of Stenson's papilla are frequently curative in the early stage of the disease Later, when suppuration, sacculation, and scarring have become extensive, surgical excision of the parotid gland (total parotidectomy) is indicated. The facial nerve must be preserved.

CHRONIC INFLAMMATION OF SUBMAXILLARY GLANDS

Chronic inflammation of the submaxillary gland occurs secondary to calculus or stricture of the duct system. The process continues for years until the gland becomes permanently enlarged by infiltration of lymphocytes and formation of scar tissue.

Probing of the duct with a lacrimal duct probe usually aids in the diagnosis of stricture or stone, or both. X-rays may show stones. Sialography may aid in the diagnosis.

Conservative treatment should be tried early: duct dilatation, removal of stones near the papilla of Wharton's duct, and meatotomy of the duct papilla.

In the advanced case where there is permanent damage to the gland with recurrent pain and suppuration, the gland itself should be removed surgically.

SALIVARY CALCULI

Salivary calculi occur more frequently in the submaxillary duct (Wharton's) than in the ducts of the other salivary glands (parotid

*Thyroiditis is discussed on p. 192.

and sublingual). There may be single or multiple calculi or "grav-el." The calculi are composed of inorganic salts (primarily calcium and phosphorus), and often have a central core of foreign substance. Calculi vary in size from 1 mm. to several cm.

Symptoms and signs produced by calculi depend upon their size and location. Obstruction of Wharton's duct causes swelling of the submaxillary gland and pain, often after eating. Symptoms and signs of acute inflammation may be most prominent.

Palpation of the calculus or calculi in Wharton's duct in the floor of the mouth is diagnostic   Stones in the hilum of the gland area can usually be felt only with bimanual palpation.

Opaque calculi are readily discernible in the x-ray film.

If symptoms and signs of acute inflammation are present, con-servative care should be instituted   When the calculus is near the papilla of Wharton's duct, the papilla may be cut off or slit and the stone evacuated. Some stones in this area may be "milked out" of the duct after dilatation with a lacrimal duct dilator.

The intraoral surgical removal of calculi from Wharton's duct is feasible provided the calculi are not too far back in the duct. A probe should be inserted into the duct as a guide for surgical in-cision. After removing the calculus, the slit duct need not be re-paired.

Calculi in the distal third of Wharton's duct and those in the hilum of the gland should not be removed intraorally. The treat-ment of choice in this instance is to remove the gland and stone by an external surgical approach, ligating the oral stump of the duct.

TUBERCULOSIS OF THE HEAD AND NECK

Cervical Lymph Node Tuberculosis.

Tuberculosis of the cervical lymph nodes may be secondary to a tuberculous focus in the gums or tonsils or may originate in dis-tant sources (e.g., the lungs, by miliary spread). The superior cervical nodes are usually involved first, and the lower nodes of the neck later through lymphatic pathways. The inflammation in the nodes goes through an initial stage of exudation, followed by caseation and liquefaction. Sinuses to the surface may develop, as in scrofula. Pus from the draining sinuses may allow identification of Mycobacterium tuberculosis. In arrested cases x-rays may show calcifications within the cervical lymph nodes.

In early cervical lymph node tuberculosis, the patient should be at bed rest, and measures to promote improvement in general health should be instituted as well as antituberculosis chemotherapy. Observation will usually determine whether the process will re-solve or progress. If conservative measures fail, infected nodes should be excised. Antituberculosis chemotherapy should be used as an adjunct to surgical therapy.

Laryngeal Tuberculosis.

Laryngeal tuberculosis follows active pulmonary disease. It must be considered in the differential diagnosis of carcinoma and gummas of the larynx. The diagnosis is usually made by biopsy and microscopic tissue examination. Treatment consists of voice rest and the use of antituberculosis chemotherapy. Tracheostomy has rarely been indicated in recent years.

ACUTE CERVICAL ADENITIS
(With or Without Abscess)

Acute inflammatory adenitis is the most common lesion occurring in the neck in children. The anterior or posterior cervical nodes may be involved, depending upon whether the source of inflammation is in the scalp or ear or in the nasal or oral cavity. Cervical lymph gland inflammation is frequently seen with acute or chronic tonsillitis. Symptoms and signs depend upon the virulence of the infection, its location and source, and whether suppuration develops. Many inflammatory processes of the cervical nodes resolve spontaneously when the primary source is eradicated.

In those cases where suppuration and breakdown of lymph nodes occurs, and when fluctuation is evident, incision and drainage are indicated. Superficial abscesses can be entered by sharp incision or with an aspirating needle. The deeper abscesses must be entered by blunt dissection after the skin and platysmus muscle are cleanly incised. If the central mass of the abscessed lymph node is entered and drained (Penrose drain for 3-5 days), resolution usually takes place.

INFECTIONS OF THE ORAL CAVITY

PERITONSILLAR ABSCESS

Peritonsillar abscess is a complication of acute tonsillitis. The infection spreads to the potential anatomic peritonsillar space adjacent to the tonsil bed and soft tissues of the palate. Mixed pyogenic organisms (streptococci, staphylococci, and/or pneumococci) are usually obtained upon culture.

The patient complains of sore throat on 1 side. Pain is severe on swallowing. Trismus is present. Upon inspection there is unilateral swelling which pushes the tonsil toward or across the midline. The swelling extends to the soft palate. The uvula is displaced. Cervical adenitis may be evident. Fluctuation develops between the third and fifth days.

Symptomatic care and antibiotic treatment are indicated early. Incision and drainage of the abscess are indicated after fluctuation develops (3-5 days). Important after-care consists of spreading the abscess cavity walls daily to avoid re-formation of the abscess. Tonsillectomy is usually indicated approximately 1 month after clearing of the peritonsillar abscess.

LUDWIG'S ANGINA

This is a severe mixed pyogenic infection of the submaxillary and sublingual fascial spaces of the floor of the mouth and upper neck. There is a rapidly spreading, diffuse cellulitis of the submental areas, with limitation of motion of the mandible and tongue. The tongue may be immobilized against the roof of the mouth. The

airway may become obstructed. Edema and painful swelling are noted in the submental areas and the floor of the mouth.

Treatment consists of massive doses of antibiotic drugs and supportive measures. If the condition progresses, incision and drainage should be done externally. Local anesthesia must be used to avoid the danger of immediate obstruction of the airway, which may occur if general anesthesia is used. Because of the diffuse nature of the infection, large quantities of free pus are seldom obtained. Incision must be adequate, and the fascial spaces above and below the hyoglossus muscles must be opened by blunt dissection.

Caution: Be prepared to perform a tracheostomy in these patients (see p. 166).

RETROPHARYNGEAL ABSCESS

This pyogenic infection occurs most frequently in infants and children. Suppuration occurs in the areolar tissue spaces between the posterior pharyngeal wall and the prevertebral fascia as a result of suppurative lymphadenitis. The patient complains of difficulty in swallowing or breathing, or both. Signs of sepsis are present. Examination of the oropharynx will show a fluctuant swelling of the posterior pharyngeal wall.

Supportive measures (antibiotics, hydration, and proper nourishment) should be instituted. When incision and drainage are required, the patient should be positioned in the deep Trendelenburg position. Adequate lighting and suction are essential, and the physician must be prepared to do a tracheostomy if indicated. General anesthesia is contraindicated because of the danger of laryngeal obstruction and aspiration of pus and blood.

PARAPHARYNGEAL ABSCESS

Parapharyngeal space abscess is a pyogenic infection secondary to acute tonsillitis, peritonsillar abscess, dental infections, or acute pharyngitis. This potential anatomic space lies in close relation to the superior and middle pharyngeal constrictor muscles, the stylopharyngeus and stylohyoideus muscles, and the carotid sheath. Infection can gravitate along the carotid sheath to the mediastinum. There are symptoms and signs of sepsis with trismus, and bulging of the lateral pharyngeal wall. The veins of the neck and scalp may be distended from jugular vein pressure. In more advanced suppurations of this space, brawny swelling, edema, and redness may develop in the neck below the angle of the mandible.

Supportive measures are indicated early (hydration, antibiotics). Intraoral incision and drainage should be done only by a surgeon experienced in operations in this area because of the danger of hemorrhage from large blood vessels. External incision and drainage at the angle of the jaw and upper neck area can be done if pus is sought deep within the neck by blunt dissection.

Caution is required in giving these patients general anesthesia, since the laryngeal airway may be closed off by sudden edema even

before intratracheal intubation can be done. Local anesthesia should be used or a tracheostomy considered.

INFECTIONS OF
THE EAR AND MASTOID BONE

PERICHONDRITIS AND CHONDRITIS OF THE EAR

Perichondritis of the cartilage of the pinna is important because of the "cauliflower" deformity which may result. Infection is usually due to pyogenic organisms and may follow trauma, surgery, insect bites, or frostbite, or may occur spontaneously. If the perichondrium is lifted from the cartilage, chondritis develops. The pinna becomes swollen, reddened, and edematous. Treatment is usually conservative, with streptomycin and chloramphenicol. Incision and drainage must be judiciously used.

EXTERNAL OTITIS

External otitis is a common diffuse inflammatory skin infection of the external auditory canal. It is caused by a mixed group of pyogenic organisms (staphylococci, streptococci, proteus organisms) or, less frequently, by fungi. Treatment is conservative, usually in the form of cleansing, Burow's solution compresses (1:12), and systemic antibiotics.

ACUTE SUPPURATIVE OTITIS MEDIA

This disease is most commonly seen in infants and children, but it can occur at any age. Suppuration of the middle ear usually develops following or accompanying disease of the upper respiratory tract.

Pyogenic organisms (beta-hemolytic streptocci, staphylococci, pneumococci, and H. influenzae) are the usual infecting organisms. There are symptoms and signs of inflammation, such as fever, pain, and a serosanguinous or purulent aural discharge. Examination of the ear shows a reddened, bulging tympanic membrane. There may be exudate in the external auditory canal if the tympanic membrane has ruptured spontaneously. Rarely can the perforation of the tympanic membrane be seen at this stage of the disease.

Acute mastoiditis may occur as a complication.

Systemic antibiotic therapy should be instituted. Penicillin is usually the drug of choice. Paracentesis of the tympanic membrane should be done routinely even if the tympanic membrane has ruptured spontaneously.

SEROUS OTITIS MEDIA
(Catarrhal Otitis Media)

Serous otitis media may occur at any age. It is characterized by exudation (transudation) of serum or mucus into the middle ear

cavity. In most cases no organisms can be obtained by culture of the aspirated material from the middle ear. The patient experiences a stuffiness of the ear and a conduction hearing loss. The tympanic membrane is often yellowish and retracted in appearance, whereas the ossicular chain appears chalky. In adults cancer of the nasopharynx must be ruled out in unilateral instances of serous otitis media.

Local treatment consists of eustachian tube inflations, paracentesis of the drum membrane with aspirations of the middle ear contents, and phenylephrine (Neo-Synephrine®) nose drops (0.25% solution). Systemic antihistamine drug therapy is indicated. Secondary contributing factors must be corrected, e.g., by tonsillectomy and/or adenoidectomy, control of allergic disorders of the respiratory system, and treatment of suppurative sinusitis.

CHRONIC OTITIS MEDIA

Chronic inflammatory processes of the middle ear are usually associated with either eustachian tube salpingitis or chronic mastoiditis. They follow acute suppurative otitis media (particularly that associated with acute exanthematous diseases of infancy or childhood). The tympanic membrane is perforated in 1 of 3 areas: central, marginal, or attic. Hearing is usually impaired.

Contributing factors such as tonsillitis, adenoiditis, sinusitis, and nasal polyps should be corrected. Local treatment is usually in the form of ear drops (alcohol and boric acid, or antibiotic solutions) or powders (iodine, or antibiotics alone or combined with boric acid). Saturated urea solution is an excellent cleansing agent.

If there is evidence of continued suppuration, or if complications occur, radical or modified radical mastoidectomy should be done.

MASTOIDITIS

Acute mastoiditis is a complication of acute suppurative otitis media. Bony necrosis of the mastoid process and breakdown of the bony intercullular structures occurs in the second to third week. When this happens, there is evidence of continued suppuration of the middle ear, mastoid tenderness, signs of sepsis, and x-ray evidence of bone destruction. If suppurative mastoiditis develops in spite of antibiotic therapy, complete mastoidectomy must be done. Acute mastoiditis is rarely seen since chemotherapeutic and antibiotic therapy has become universal in the treatment of acute suppurative otitis media.

Chronic mastoiditis is a complication of chronic suppurative otitis media. If the disease occurs in infancy, the mastoid bone does not develop cellular structures but becomes sclerotic. Infection is usually limited to the antral area. In some cases of marginal and attic perforations of the tympanic membrane, cholesteatomas develop. Cholesteatomas are produced by the ingrowth of squamous epithelium from the skin of the external auditory canal. By their laminated growth, they may erode adjacent bone or soft tissue.

The use of antibiotic drugs in chronic mastoiditis is usually

not helpful in clearing the mastoid infection but may be effective in the treatment of complications. Many cases of chronic otitis media and mastoiditis can be managed by local cleansing of the ear and instillation of antibiotic powders or solutions. In other cases, radical or modified radical mastoidectomy must be done.

COMPLICATIONS OF EAR INFECTIONS

Following Acute Suppurative Otitis Media and Mastoiditis.
  A. Subperiosteal abscess secondary to acute mastoiditis and suppurative acute otitis media may be encountered.
  B. Facial nerve paralysis developing in the first few hours or days after the onset of acute suppurative otitis media is due to edema about the nerve in the bony facial canal. Conservative treatment is usually indicated (antibiotic therapy, paracentesis, and supportive measures).
  C. Meningitis, abscess (epidural, subdural, and brain), and sigmoid sinus thrombosis are serious complications following suppurative otitis meaia and mastoiditis. These complications may be masked by use of the antibiotic drugs. Surgical treatment of the mastoid disease and its complications is indicated.

Following Chronic Suppurative Otitis Media.
  A. Acute exacerbations of chronic otitis media and mastoiditis may lead to meningitis, epidural and brain abscess, and sigmoid sinus thrombosis. Antibiotic therapy and surgical intervention should be carried out.
  B. Facial nerve paralysis is usually the result of direct pressure from cholesteatomas or granulation tissue. Surgical intervention is imperative.

Labyrinthitis.
  Acute suppurative labyrinthitis is an infection of the intralabyrinthine structures. It may be secondary to acute suppurative otitis media and mastoiditis, acute exacerbations of chronic suppurative otitis media and mastoiditis, or meningitis unrelated to otitis media and mastoiditis. There is usually complete destruction of labyrinthine function and total deafness. Antibiotic drugs and surgical drainage are indicated.
  Chronic labyrinthitis is secondary to erosion of the lateral bony semicircular canal by cholesteatoma. The patient is chronically dizzy. Mastoidectomy and removal of the cholesteatoma is the treatment of choice. •

INFECTIONS OF THE PARANASAL SINUSES

  The maxillary sinuses are involved in acute suppuration which follows acute upper respiratory disease, swimming, abscessed teeth, dental extractions, and nasal allergies. Pyogenic organisms are the etiologic agents. Acute inflammatory processes of the maxillary sinuses are characterized by pain and tenderness over the cheek and teeth. Pus is evident in the nose. The early treatment of acute empyema of the maxillary sinus is conservative, with the

use of hot packs, oral antihistaminic drugs, local vasoconstrictors, and control of pain by narcotic drugs if necessary. Antibiotic drugs are indicated, particularly when the patient has fever, tenderness, and pus exuding from the sinus osteum. Antral irrigations should be done later, after the acute process subsides.

Isolated infection of the frontal sinuses is rare. Treatment is usually conservative. The frontal sinus or its nasofrontal duct should rarely be cannulated. Trephining of the sinus floor may be indicated in acute fulminating infections.

Acute ethmoiditis causes swelling along the inner canthus of the eye. It occurs most frequently in children and infants. At these ages the ethmoidal sinuses are well developed. Treatment in most instances is conservative; only if fluctuation develops externally should incision and drainage be done.

Chronic suppurative sinus disease occurs principally in adults. Chronic pyogenic infections of single sinuses do occur, but are less common than pansinusitis. The patient has few symptoms except purulent rhinorrhea. Antibiotic drug therapy is usually indicated when the specific pyogenic organism is isolated. Conservative surgery (including irrigations) to promote drainage must be used whenever possible. If conservative treatment fails, more radical sinus surgery (external approach) is permissible.

The following complications of chronic suppurative sinus disease may occur: (1) Osteomyelitis of the facial or skull bones. (2) Meningitis and epidural, subdural, and brain abscess. The treatment of (1) and (2) consists of supportive measures, adequate antibiotic and chemotherapy, and surgery as indicated. (3) Draining orbital fistulas are complications of frontal or ethmoidal chronic suppurative disease. The underlying pathology of the sinus must be eliminated surgically before the fistulous tract can be closed permanently. (4) Oro-antral fistulas occur following tooth extractions, trauma (penetrating wounds, surgical procedures), bone infection, and neoplasms occurring in the area of the maxillary sinus. Chronic suppuration of the sinus frequently is present. The successful surgical treatment of the fistula is dependent for the most part on eradicating the sinus disease successfully. (5) Mucoceles (mucopyoceles) develop most frequently in the anterior ethmoidal sinuses and encroach upon the floor of the frontal sinuses by extension. They result from the blocking of sinus drainage. Proptosis of the orbital contents is common. Surgical excision of the mucocele is necessary for complete cure.

NEOPLASMS OF THE HEAD AND NECK

BENIGN NEOPLASMS OF THE ORAL CAVITY

Warts, Verrucae, and Epithelial Papillomas.

These tumors may be sessile or pedunculated. Microscopically, there is proliferation of the squamous epithelium, which grows into folds; within the stalk there is a small amount of connective tissue and blood vessels. Excision is the treatment of choice either by sharp or electrocautery dissection.
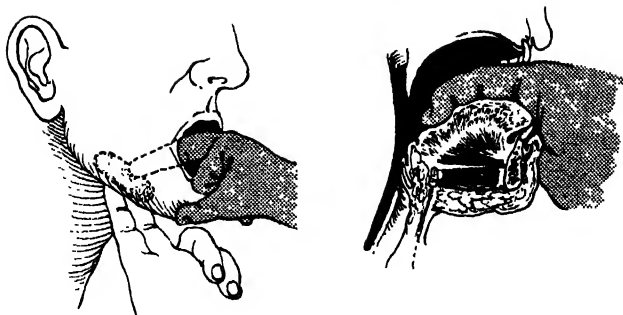
Manual Palpation of the Structures of the Floor of the Mouth

Leukoplakia.

Leukoplakia appears as whitish plaques on the tongue or palate or on the buccal surfaces of the cheeks, lips, or gingivae, and should be considered premalignant. Patients are often heavy smokers. Microscopically, leukoplakia may be likened to the hyperkeratotic lesions of the skin with the exception that in these locations there are no hair follicles or sweat and sebaceous glands. Malignant changes may occur in the basal cell layers. All suspicious lesions must be adequately biopsied.

Observation is essential to determine changes in growth. Multiple and frequent biopsies may be necessary.

Remove sources of irritation such as ill-fitting dentures, jagged teeth, and tobacco. Excise surgically if healing does not occur following conservative treatment.

Hemangioma and Lymphangioma.

Hemangiomas and lymphangiomas occur on the mucous membranes of any part of the oral cavity and are often noted soon after birth. If they occur within the tongue, "macroglossia" may result. Multiple or single lesions may be present.

Hemangiomas have a purplish pigmentation; lymphangiomas are the color of the underlying mucous membrane or paler. Microscopically, these lesions may be capillary or cavernous or mixed. The diagnosis is made by inspection and palpation and not by biopsy.

Surgical excision, whether by sharp dissection or electrocautery, is the treatment of choice if it can be done safely and there is a good chance of total excision. Radiation therapy may be effective, particularly in younger patients. Sclerosing agents (sodium morrhuate, urethane, and quinine hydrochloride) may be tried, but excessive amounts of these drugs must not be injected.

Median Rhomboid Glossitis.

Median rhomboid glossitis is considered here since it is frequently confused with neoplastic disease. It occurs as a painless, shiny, reddened, raised, firm lesion in the midline of the tongue
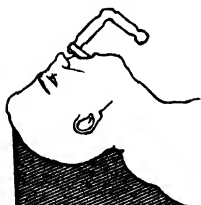
**Position of Patient for
Direct Laryngoscopy**
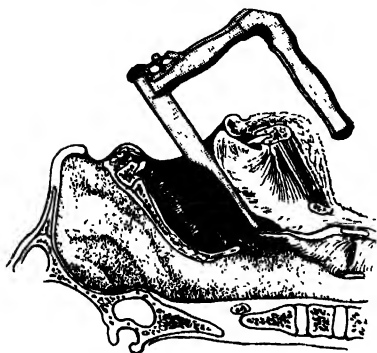
Standard Distal Lighting Laryngoscope Being Introduced. Tip of
laryngoscope in vallecula. At right, anterior surface of epiglottis
and vallecula as seen with the laryngoscope in the position shown
at left.
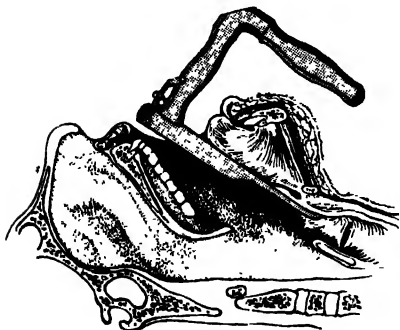
Tip of Laryngoscope Within Larynx. At right, direct view within
larynx as seen with the laryngoscope in the position shown at left.

Indirect Mirror Examination of the Larynx and Adjacent Structures

Preglandular node (drains floor of mouth, gums, buccal mucosa, ant. 2/3 of tongue)

Submental nodes (drain lip)

Pretracheal node (drains thyroid)

Middle deep cervical node (drains oral cavity, nasopharynx, larynx, scalp; 2° spread from sup. and mid. deep cervical nodes)

Inferior deep cervical node (drains thyroid and upper esophagus)

Superior deep cervical node (drains oral cavity, nasopharynx, larynx)

Spinal accessory chain (drains scalp; 2° spread from sup. and mid. deep cervical nodes)

Virchow's node (drains lesions below the clavicle)

Lymphatic Drainage in the Neck (Left Lateral View). (Modified after Richards.)

just in front of the circumvallate papillae. The cause is not known.
Microscopic examination shows chronic inflammatory changes of
the tissue. No treatment is necessary, and biopsy is not indicated.

Aberrant Salivary Gland Tumors.

Aberrant salivary gland tumors may occur in the cheeks, the
palate, or the pharynx. They arise from epithelial salivary gland
rests or from epithelial metaplasia of secondary salivary glands.
There are usually few or no symptoms. The surface is covered
with intact epithelium. About 30% of these tumors undergo malig-
nant changes.

Treatment is by complete surgical excision.

Palatal Exostosis (Torus Palatinus).

An exostosis is frequently seen in the bony palate. It occurs as
a painless bony swelling in the midline covered by intact mucosa.
Exostoses are usually found on routine examination. No treatment
is required unless the process interferes with the wearing of arti-
ficial dentures.

MALIGNANT NEOPLASMS OF THE ORAL CAVITY

Squamous Cell Carcinoma.

Squamous cell carcinoma is the most common malignant neo-
plasm in the oral cavity. (Adenocarcinoma is rare.) It may arise
in areas of leukoplakia. On the mucous membrane surfaces it
appears as ulcerations with elevated borders. Infiltration and in-
duration may be evident, or the growth may be papillary, or both.
The lesions tend to spread by direct extension and through the
lymphatic vessels to regional lymph nodes or, less commonly, via
the blood stream. Every ulceration of the oral cavity should be
biopsied promptly.

A. Palate and Gingivae: Squamous cell carcinoma may arise on
   the hard or soft palate or gingivae. If it originates on the sur-
   face of the hard palate, erosion into the nasal cavity or into
   the maxillary sinus may be evident. Lesions of the soft palate
   arise on the dorsal or ventral surface or on the uvula. If they
   arise on the dorsum, early clinical diagnosis can be made only
   by mirror or nasopharyngoscopic examination of the area.
   Definitive diagnosis is made by microscopic examination of bi-
   opsy specimens. Pain is the most frequent complaint. X-rays
   should be obtained to rule out bone invasion.

   Small lesions may be excised if they are surrounded by a
   wide margin of normal tissue. More extensive lesions can be
   treated with radiation or sharp and electrocautery excision, or
   both. If bone is invaded, surgical excision is the treatment of
   choice. Regional neck lymph node metastases should be treated
   by radical neck dissection only if the primary lesion has been
   treated successfully.

B. Buccal Mucosa: Squamous cell carcinoma of the buccal mucosa
   usually arises in an area of leukoplakia. Small lesions can be
   treated by surgical excision or radiation therapy; more exten-
   sive lesions may require both. Regional lymph node metastases
   are treated by surgical excision (block dissection of the neck if

the primary lesion is controlled). If there is extensive loss of tissue following treatment, plastic repair in the form of pedicle grafting may have to be carried out.

C. Floor of Mouth: Squamous cell carcinoma arising in the floor of the mouth may involve a portion of the tongue or mandible by direct extension. Because the area contains abundant lymphatic channels, early metastasis to submental, submaxillary, and jugular lymph nodes on either side is the rule. Diagnosis is made by inspection, palpation, and microscopic examination of biopsy specimens. Bimanual palpation is important to determine the extent of infiltration into the tissues of the floor of the mouth. X-rays should be obtained to rule out bone involvement of the mandible.

Small primary lesions can be excised (scalpel or electro-dissection) intraorally; some clinicians use radiation therapy also. More extensive lesions require surgical excision by a combination of the intraoral and suprahyoid routes ("pull-through operation"). If the lesion has involved the periosteum or the bone of the mandible, a section of the mandible must be excised in continuity with the primary growth. A section of the tongue may have to be excised as well. Block dissection of the neck is indicated if the primary lesion is arrested and if resectable nodes are present in the neck, and in patients with extensive primary lesions as a prophylactic measure.

D. Tongue: Squamous cell carcinoma may occur on the body, tip, sides, under-surface, and posterior surface of the tongue. The tongue is rich in lymphatic channels, and these lesions tend to metastasize early. Pain is the most common symptom. Palpation reveals the extent of local infiltration and involvement of the lymph nodes of the neck. The diagnosis is confirmed by microscopic examination of biopsy specimens.

The treatment of squamous cell carcinoma of the tongue depends upon the location of the primary lesion and its potential or actual lymph node extensions. Small cancers of the tip are ideally located for wedge-shaped excision of the tip of the tongue. Cancer of the body of the tongue must be excised widely. Hemiglossectomy combined with radical neck dissection is the treatment of choice. Primary cancer of the posterior third of the tongue has a poor prognosis regardless of treatment. Partial glossectomy combined with radical neck dissection and/or radiation has been used.

If neck nodes develop after treatment of the primary lesion and it is reasonably certain that the primary lesion has been arrested, radical neck dissection should be done.

Intraoral and external x-ray therapy have been used successfully in some cases.

BENIGN NEOPLASMS OF THE LIPS

Hyperkeratosis.

Hyperkeratosis of the upper or lower lip often develops in persons whose occupations keep them out-of-doors. These lesions are considered premalignant. Leukoplakia and hyperkeratosis may be

adjacent lesions, the former occurring on the inner surface of the
lip and the latter on the vermilion border. Hyperkeratosis may
appear as a discrete area, or the whole lip may be involved along
its vermilion border. Microscopically, the stratified epithelium
becomes thickened and hypertrophied, and excessive keratin layers
are formed and shed so that the surface appears cornified and scaly.
The rete pegs are elongated, but the basement membrane is intact.
Malignant change "in situ" or invasive carcinoma may be evident.
Induration as noted by palpation is suggestive of malignant change.
Biopsy or biopsy excision (small lesions) should be done for defini-
tive microscopic diagnosis.

In cases showing early hyperkeratosis, the application of pro-
tective ointments may be sufficient to promote healing. Slightly
larger areas may be treated by electrodesiccation if malignancy
has been ruled out by biopsy. Such coagulated areas should be pro-
tected by bland oils, ointments, or 2% aqueous gentian violet appli-
cations.

When the entire lip is involved, the vermilion surface is ex-
cised and the buccal mucosa undermined, advanced, and sutured to
cover the defect.

MALIGNANT NEOPLASMS OF THE LIPS

Squamous Cell Carcinoma.

Squamous cell carcinoma of the lips occurs spontaneously in
areas of hyperkeratosis (leukoplakia) and in ulcerations. The lower
lip is more frequently involved, and the lesion is more common in
men than in women. Sunlight appears to be a predisposing factor.
The lesion appears as a raised, ulcerated area which cracks and
bleeds easily on manipulation. Induration is evident upon palpation.
In all lesions the submental areas and neck should be palpated to
determine lymph node involvement. Diagnosis is confirmed by
microscopic examination of suitable tissue.

Lesions 1.5-2 cm. in diameter which do not infiltrate deeply
may be excised by removing a full-thickness wedge or V of the lip.
Suprahyoid node dissection is not indicated. External radiation
therapy is sometimes used.

Larger lesions require more extensive local excision and often
suprahyoid and radical neck dissection as well.

If the primary lesion is controlled and the nodes appear in the
neck at a later date, radical neck dissection should be done.

BENIGN NEOPLASMS OF THE NASAL CHAMBERS
AND PARANASAL SINUSES

Squamous Papillomas (Inverting Papillomas).

Squamous cell papillomas arise either in the paranasal sinuses
or in the nasal chambers. They are benign lesions in the sense that
they do not metastasize; however, they erode adjacent structures

(soft tissue and bone) by their local growth. Grossly, the papillomas appear irregular on the surface and firm in character, often filling a whole nasal chamber. Histologically, they show squamous epithelium growing in papilliferous cords surrounding a fibrous core. Definitive diagnosis is made by microscopic tissue examination. X-rays should be taken to identify the sinuses involved and to determine if bony erosion has occurred.

Complete removal often requires radical surgery, to the point of eradicating the paranasal sinuses and, at times, the orbital contents as well.

Hemangiomas.

Hemangiomas in the nose or paranasal sinuses are diagnosed by a history of epistaxis and the finding of the characteristic bluish tumor. Treatment is usually by radiation therapy in combination with surgical excision.

Osteomas and Ossifying Fibromas.

Osteomas occur characteristically in the maxillary and frontal sinuses. They usually cause no symptoms unless they grow to excessive size. Many are diagnosed only by routine x-rays. No treatment is indicated unless symptoms are present.

Ossifying fibromas may occur in the maxillary sinus and upper jaw. These benign tumors contain bone as well as fibrous tissue in varying amounts. They grow slowly and may thin adjacent bone by their expansive growth. Symptoms depend on the size of the lesion and involvement of neighboring structures. X-rays are an aid in diagnosis. Treatment is by surgical excision, which must be extensive if the palate or orbital bony structures are involved.

Nasal Polyps.

Nasal polyps are not neoplastic in origin but are considered here because they are often confused with neoplastic lesions. They are associated with allergy and infection or both. Treatment is by surgical removal. All excised tissues, regardless of their gross appearance, should be examined microscopically.

MALIGNANT NEOPLASMS OF THE NASAL CHAMBERS AND PARANASAL SINUSES

Squamous Cell Carcinoma.

Squamous cell carcinoma can develop in the nose or in any of the paranasal sinuses. It occurs most frequently in the maxillary antrum. The sinus mucosa undergoes metaplasia and malignant changes. Microscopic examination of the lesion shows squamous cell carcinoma of varying degrees of differentiation. Symptoms and signs often appear late in the disease and depend upon the extent of the lesion, its speed of growth, the nerves involved, and the presence of infection and bone involvement. All tumors of the nasal chambers or paranasal sinuses which are not obviously benign should be considered carcinoma until proved otherwise. Biopsy and microscopic tissue examination will confirm the diagnosis. Spread is by direct extension to adjacent structures; via lymphatic channels to regional lymph nodes in the pterygomaxillary space,

submandibular nodes, and cervical nodes; and, infrequently, via the blood stream.

Surgical excision is difficult because these tumors grow so extensively. However, whenever feasible this is the treatment of choice. The neoplasm is resected by an external approach.

Surgery and radiation therapy are often combined.

If the primary neoplasm is arrested and resectable cervical metastases are present, radical neck dissection should be done.

BENIGN NEOPLASMS OF THE JAW

Adamantinomas (Ameloblastomas).

Adamantinomas occur more frequently in the lower than in the upper jaw. They grow by expansion, causing a swelling intraorally or externally (or both). They arise from the enamel organ epithelium of a tooth. The tumor itself contains cystic spaces which are filled with colloid, mucinous, or clear yellow fluid. Microscopically, the cystic spaces are surrounded by bony trabeculae that contain cuboidal stellate or tall columnar epithelium alone or in combination. Some tumors may be predominantly cellular. They metastasize rarely to distant long bones, lungs, and regional lymph nodes. Symptoms and signs are those of a slowly growing, painless mass in the upper or lower jaw. As the cortex of the jaw is thinned, palpation may elicit a sense of crackling. X-rays aid in the diagnosis if the cystic areas are a prominent feature of the lesion. Biopsy, usually through an intact mucosa or intact skin surface, and microscopic tissue examinations are essential for final diagnosis.

Surgical excision of small lesions of the lower or upper jaw usually present no difficulties. However, large tumors may require extensive resection of the mandible or maxilla and palate. Complete surgical excision of the lesion must be done if local recurrences are to be prevented.

Giant Cell Tumor.

Giant cell tumors of the mandible are not unlike those found elsewhere in the body. Treatment is by surgical excision, which can usually be accomplished without interrupting the continuity of the jaw.

MALIGNANT NEOPLASMS OF THE JAW

Sarcoma is the most common of the rare primary malignant tumors of the jaw. Metastatic tumors may invade the jaw, or the jaw may be involved by direct extension from neighboring neoplasms X-rays may aid in diagnosis. A diligent search for the primary lesion must be made elsewhere in the body.

BENIGN NEOPLASMS OF THE NASOPHARYNX

Juvenile Fibroma (Juvenile Nasopharyngeal Angiofibroma).

Juvenile fibromas occur most frequently in the adolescent male. They arise in or near the vault of the nasopharynx and may reach

considerable size. They are attached to the underlying structures by a stalk or by a sessile base. Histologically, the tumor is composed of vascular connective tissue. The vascularity of individual tumors varies considerably.

These tumors do not metastasize. Nasal obstruction is a prominent symptom, but nasal hemorrhage is not. The tumor mass may be seen by elevation of the soft palate, indirect mirror examination of the nasopharynx, or by inspection through the nasal chambers. On finger palpation the tumor is firm and smooth. Biopsy should not be attempted unless complete hospital surgical facilities are available. X-rays reveal a soft tissue mass in the nasopharynx.

Surgical removal is the treatment of choice, either intraorally or externally (Fergusson's approach). An attempt is made to evulse the lesion at its base. Hemorrhage may be a serious complication.

Some surgeons prefer to use radiation therapy before surgery in order to reduce the tumor's vascularity.

SQUAMOUS CELL CARCINOMA OF THE NASOPHARYNX
(Lympho-epithelioma)

Squamous cell carcinoma of the nasopharynx arises in any part of the nasopharynx. Histologically, the cells may be so anaplastic that it is difficult to determine their type. Lymphoid tissue can be abundant in the stroma. Invasion of the base of the skull by direct extension and neck lymph node metastases often occurs early. There are few symptoms and signs until later in the disease, when epistaxis, pain, unilateral serous otitis media, a mass in the neck, and cranial nerve involvement may occur. There is a rather high incidence of this neoplasm in Orientals. The diagnosis is made by microscopic examination of representative tissue specimens. X-rays are necessary to determine bony involvement of the base of the skull and invasion of the paranasal sinuses.

External x-ray therapy is the primary treatment of choice. Recurrences are treated by the insertion of radioactive cobalt or radium into the nasopharynx. (The nasal septum may first have to be resected.)

Cervical metastases are treated by radiation therapy. Radical neck dissection is rarely indicated because of the anaplastic nature of the tumor.

SQUAMOUS CELL CARCINOMA OF THE TONSILS

Squamous cell carcinoma of the tonsils accounts for about 10% of all intraoral carcinomas. Microscopically, the cell type may vary considerably from the epidermoid to the transitional cell type. The symptoms and signs depend on the size of the lesion, ulceration, infiltration, and secondary infection. Pain is an outstanding symptom. The exact origin of the primary lesion is often difficult to determine if the process has spread to the tongue, pillars, and soft palate. Diagnosis is confirmed by microscopic examination of representative tissue. Since lymph channels are abundant in this

area, neck lymph node involvement occurs early.

Surgical excision of squamous cell carcinomas arising in the tonsils should be radical. In order to remove the lesion and its regional lymph nodes, radical neck dissection in continuity with excision of the primary lesion must be done. To accomplish this the mandible must be partially resected or bisected.

Radiation therapy is regarded as the treatment of choice by some clinicians. Surgery and radiation therapy may be combined.

BENIGN NEOPLASMS OF THE LARYNX AND HYPOPHARYNX

Polyps, papillomas, vocal nodules, and leukoplakia may occur on the vocal cords, producing hoarseness. Diagnosis is made by direct and indirect laryngoscopy and biopsy (often biopsy excision). The lesions are treated by excision except for the vocal nodules, which may be treated by voice training to prevent misuse and over-use of the voice. Papillomas and leukoplakia must be considered premalignant lesions.

MALIGNANT NEOPLASMS OF THE LARYNX AND HYPOPHARYNX

Squamous Cell Carcinoma.

Squamous cell carcinomas represent 99% of all malignant tumors of the larynx. About 55-65% originate on the true vocal cord; the rest in the laryngeal ventricles, false cords, aryepiglottic folds, epiglottis, and arytenoid and subglottic areas. The sites of primary malignancy in the pharynx are the pyriform sinuses, lateral pharyngeal walls, and postcricoid areas. Histologically, the lesions of the true cord are more differentiated than those of other locations. They are characteristically of the epidermoid type.

Hoarseness is an early symptom of true vocal cord cancer. Laryngeal cancers of other areas are less apt to produce hoarseness, but cause pain and dysphagia. Biopsy (indirect or direct) confirms the diagnosis.

True vocal cord cancer metastasizes late. Cancers arising in other locations metastasize earlier and more frequently because of their anaplastic cell type and the abundant lymph channels in these areas. The lymph nodes involved are along the internal jugular vein; those overlying the thyrohyoid and cricothyroid membranes; and those in the potential anatomic space between the esophagus and trachea. Other routes of spread of laryngeal cancer are by direct extension and, rarely, via the blood stream.

Peroral forceps removal is the treatment of choice in small (2 mm.) true vocal cord cancer. Electrocautery of the base is a further safeguard.

True vocal cord lesions involving the middle third of the cord may be removed by splitting the thyroid cartilage and removing part of the false and the entire true vocal cord (laryngofissure).

More extensive lesions of the true vocal cord require either hemilaryngectomy or wide-field total laryngectomy. True vocal cord lesions require radical neck dissection in continuity with the

laryngectomy if nodes are palpable in the neck or if the primary lesion has invaded supraglottic or infraglottic structures. In the latter instances, the node dissection is done as a prophylactic measure.

As a rule those cancers arising in other locations in the larynx or pharynx require wide-field laryngectomy alone (infrequent) or wide-field laryngectomy combined with ipsilateral radical neck dissection. Contralateral neck dissection may be indicated also, but usually at a later date. Prophylactic ipsilateral neck dissection must be done in many cases because of the high incidence (40%) of metastatic nodes found on microscopic examination of surgical specimens.

Small cancers of the true vocal cord may also be treated by external x-ray therapy. Larger cancers of the true vocal cord or those arising in other areas carry a less favorable prognosis with this form of therapy.

Surgery combined with preoperative or postoperative x-ray therapy may be an important form of treatment in certain cases. The surgeon and radiologist must decide and administer the treatment cooperatively.

BENIGN NEOPLASMS OF THE SALIVARY GLANDS

Mixed Tumors.

About 70-80% of tumors arising in the salivary glands are of the mixed cell type. About 15% of these arise either in the submaxillary or sublingual glands; mixed tumors of the parotid gland account for the remainder. The tumors are encapsulated, but may have finger-like projections which protrude between the lobulations of the gland. Histologically, the tumors are made up of epithelial elements interspersed with fibrous (mesoblastic) tissue. The epithelial elements (alveoli, masses of cells or strands) may predominate in 1 portion of the tumor, whereas in another the mesoblastic element may predominate in the form of cartilage, myxomatous connective tissue, and hyalin.

A painless swelling develops in 1 of the salivary glands. (In the parotid gland the lower pole is a frequent site.) Occasionally these tumors arise in the deep portion of the parotid gland and their main growth is inward, toward the pharynx and palate. Tumors of the submaxillary gland present in the submaxillary triangle area. Although they grow slowly, they may reach several cm. in size. The finding of a discrete, firm, painless mass in these areas suggests the possibility of mixed tumor. Sialograms may show encroachment of the tumor upon the ductal system of the gland. Biopsy should not be done because of the danger of seeding uninvolved tissues.

Treatment of mixed tumors of the parotid gland is by surgical excision well beyond the tumor capsule and through normal gland tissues.

The facial nerve must be preserved. Before definitive surgery of the tumor is attempted the facial nerve must be identified, either peripherally or near its main trunk. As a rule, the parotid gland lateral to the facial nerve is excised with the superficially placed larger tumors. Dissection of tumors deep to the facial

nerve is much more difficult, and the tumor may have to be re-moved in sections.

Benign mixed tumors of the submaxillary gland are removed by complete excision of the gland. The mandibular division of the facial nerve must be preserved.

Mixed tumors may show malignant changes, as evidenced by metaplasia, invasion of surrounding structures, rapid growth, fixation, recurrence after incomplete removal, lymph node metas-tasis, and facial nerve paralysis. The treatment is wide surgical excision. If the facial nerve must be sacrificed, primary grafting of the nerve should be considered.

Papillary Cystadenoma Lymphomatosum (Adenolymphoma, Warthin's Tumor).

These tumors usually arise in the inferior pole of the parotid gland. They are composed of lymphocytoid tissues surrounding spaces lined with epithelium (acidophilic cells). They present as benign painless swellings, usually in older patients, and upon pal-pation appear cystic. Treatment is by surgical excision, taking care to preserve the facial nerve.

MALIGNANT NEOPLASMS OF THE SALIVARY GLANDS

Malignant disease of the salivary glands develops as a firm mass in the substance of the gland affected. Ulceration or infiltra-tion of the skin surface may occur, and adjacent structures such as muscle, other soft tissue, and bone may be invaded by malignant cells. The facial nerve is frequently paralyzed. Definitive diag-nosis depends upon microscopic examination of the tumor specimen. Late metastases occur to the lungs, abdominal viscera, bone, and brain.

Squamous cell carcinomas in the salivary glands are highly malignant. Spread is rapid, and the prognosis for life is extremely poor.

Adenocarcinomas are not necessarily limited to the salivary glands but may occur in other locations as well, e.g., the oral cavity, nasopharynx, paranasal sinuses, pharynx, and trachea. They occur with equal frequency in the parotid and submaxillary glands. Unlike the squamous cell varieties, these tumors are only occasionally rapidly fatal. They metastasize late.

Muco-epidermoid carcinomas (papillary cystic tumors) are ductal in origin. Their degree of malignancy depends principally upon the type of cell structure which predominates. Highly malig-nant tumors are composed mainly of mucus-secreting cells; in tumors of low-grade malignancy the epidermoid and intermediate cells predominate.

Malignant mixed tumors of the salivary glands have their or-igin in benign mixed tumors. They may be highly anaplastic in their growth, or a particular form of cellular malignancy may pre-dominate, i.e., they are pleomorphic.

The treatment of malignancies of the salivary glands is usually surgical, since these neoplasms are for the most part resistant to radiation therapy. Surgical excision must be extensive, with wide local excision of soft and bony tissues and radical neck dissection.

In parotid malignancies, the facial nerve usually must be sacrificed.

CAROTID BODY TUMOR
(Chemotectoma)

Carotid body tumors arise in the potential anatomic space in the bifurcation of the carotid artery in the nonchromaffin paraganglia. Microscopically the tumors show 2 main types of cellular structure: (1) large cell (alveolar) polyhedral and (2) small cell (peritheliomatous). Vascular tissue may permeate the fibrous tissue stroma so that the whole tumor appears as a dark, blood-red mass.

Clinically, these tumors grow slowly. They may be so vascular that a bruit can be noted by auscultation. Upon palpation they are shown to be fixed to the underlying structures. Carotid body tumors must be considered in the differential diagnosis in all tumor masses of the upper lateral neck. Biopsy is usually contraindicated.

Surgical excision is the treatment of choice. It can usually be done readily if the tumor is not attached to the great vessels. On the other hand, if the tumor is attached to the great vessels, removal may only be accomplished by sacrificing the vessels and by arterial by-pass or graft.

DISEASES OF THE THYROID

ECTOPIC THYROID

The thyroid gland arises embryologically as a midline diverticulum from the pharynx. In normal development it passes downward to its bilobed adult position, but in a rare case it may fail to evolve 2 lobes and is then seen in the neck as a single mass of tissue.

There are no symptoms. A round, smooth, firm, nontender mass is palpable in the midline of the neck below the hyoid bone. The mass is not attached to the skin and moves with swallowing.

Thyroglossal duct cyst is easily mistaken for ectopic thyroid. If there is any question about the diagnosis, a tracer dose of radioiodine will indicate the presence of functioning thyroid tissue.

No treatment is required. Inadvertent excision during surgery for thyroglossal duct cyst may result in hypothyroidism. When ectopic thyroid is encountered during neck surgery for some other disorder, the cosmetic appearance can be improved by dividing the mass in its midline (only if blood vessels enter from both sides) and turning both halves laterally for placement under adjacent muscles (Gross).

NONTOXIC NODULAR GOITER
(Adenomatous Goiter)

The major cause of adenomatous goiter is iodine deficiency, which results in hyperplasia, generalized enlargement of the gland, and, frequently, gross nodule formation. The process is not neo-

plastic, but when only 1 nodule is palpable it is usually impossible to distinguish nodular goiter from adenoma or carcinoma except by exploration and biopsy. The incidence of carcinoma in multinodular goiter is uncertain, but is probably about 1%.

Women are most commonly affected. There are usually no symptoms unless the gland is large enough to produce pressure on the trachea or esophagus. Substernal extension occurs occasionally and appears as a superior mediastinal mass on x-ray. The goiter may have been present for years. Diffuse thyroid enlargement may develop in females during puberty or pregnancy, when iodine requirements normally increase markedly. Hyperthyroidism is the most frequent complication.

It is impractical and unnecessary to advise surgical removal in every case of nodular goiter. The chief indications for surgical removal are (1) hyperthyroidism, (2) pressure symptoms, (3) substernal extension, (4) progressive enlargement, (5) suspicion of malignancy, and (6) cosmetic reasons.

Medical measures are of no value in long-standing nodular goiter. In the diffuse type of goiter of recent development (e.g., during puberty or pregnancy), regression may occur following administration of Thyroid, U.S.P., 60-120 mg. (1-2 gr.) daily, and Potassium Iodide Solution, N.F. (saturated), or Strong Iodine Solution, U.S.P. (Lugol's Solution), 5 drops daily in one-half glass of water. Continue thyroid and iodine therapy as long as the goiter is regressing; then advise indefinite use of iodized table salt.

HYPERTHYROIDISM

Hyperthyroidism is characterized by excessive secretion of thyroid hormone, probably as a result of stimulation of the thyroid by anterior pituitary thyrotropic hormone. Two forms are distinguished: (1) exophthalmic goiter or Graves' disease, and (2) toxic nodular goiter.

Exophthalmic goiter is usually acute in onset and is associated with diffuse thyroid enlargement. It is 4 times as common in women as in men and typically occurs between puberty and the menopause. Exophthalmos is present in most cases, probably as a result of excessive secretion of thyrotropic hormone or a closely related anterior pituitary hormone. Exophthalmos is infrequently progressive, and even more rarely unilateral.

Toxic nodular goiter is the result of the development of hyperthyroidism in a preexisting nontoxic nodular goiter. Women in the older age group are usually affected. The onset tends to be insidious, and signs of cardiac insufficiency often dominate the clinical picture to such an extent that the hyperthyroidism is overlooked. Exophthalmos is not present.

Clinical Findings.
A. Symptoms and Signs: Patients with exophthalmic goiter or toxic nodular goiter complain of nervousness, irritability, easy fatigability, increased tolerance to cold, and weight loss in spite of good appetite and food intake. The goiter is diffuse and smooth in Graves' disease and nodular in toxic nodular goiter; other signs in both conditions include a warm moist

skin, fine tremors in the hands, restlessness, quick move-
ments, and evidence of weight loss. In Graves' disease there
are exophthalmos, stare, and lid lag. Tachycardia is the
most frequent cardiovascular finding in both diseases. Atrial
fibrillation and congestive failure are not uncommon in older
patients.

B. Laboratory Findings:

1. BMR is usually elevated. A BMR of −5% or below practicall
   excludes hyperthyroidism.
2. PBI values above 8 mg. /100 ml. are suggestive of hyper-
   thyroidism.
3. Radioactive iodine uptake is increased. Erythrocyte uptake
   of I^{131} -ʟ-triiodothyronine in vitro is usually increased.
4. The blood cholesterol level may be low.

Treatment.

A. Medical Measures:

1. Radioactive iodine therapy is the treatment of choice in pa-
   tients over 40 with Graves' disease; in recurrent thyrotoxi-
   cosis after thyroidectomy; and when surgical treatment is
   refused or is contraindicated. Since irradiation therapy
   may favor malignant change, it is probably unwise to ad-
   minister a therapeutic dose of radioactive iodine to patients
   with a life expectancy of over 25 years except when surgery
   is unduly hazardous.
2. Antithyroid drugs - Thiouracil or thiocyanate preparations
   may be used as definitive therapy in young patients who re-
   fuse surgery, but the course of treatment is prolonged (up
   to 2 years) and costly and relapses are frequent (about 50%).
   The major value of antithyroid drugs is in preparation for
   operation.

B. Surgical Treatment: Subtotal thyroidectomy is the treatment
   of choice in toxic nodular goiter and in Graves' disease in pa-
   tients under 40 years of age. Preoperatively, it is desirable
   to render the patient euthyroid with an antithyroid drug and
   iodine. Surgery is not urgent. The BMR should be normal,
   signs of toxicity absent, resting pulse normal, and weight gain
   established. Rest, nutritional support, and mild barbiturate
   sedation are helpful adjunctive measures. Medical prepara-
   tion for surgery should be carried out as follows:

1. Propylthiouracil, U.S.P., 100-200 mg. q.i.d. A larger
   dose may occasionally be necessary to obtain an adequate
   response. BMR usually falls at the rate of about 1% per
   day, so that preparation requires 4-6 weeks. Occasional
   toxic reactions to propylthiouracil include granulocytopenia,
   drug fever, and rash. The patient should be examined
   weekly and a blood count obtained; if the WBC falls below
   4500 or if there are fewer than 45% granulocytes, the drug
   must be discontinued.

   Methimazole, U.S.P. (Tapazole®), 10-15 mg. q.i.d.,
   may be used in place of propylthiouracil.
2. Iodine - Ten to 15 days prior to operation (when the BMR is
   +10 to +15%), iodine is given in addition to propylthiouracil.
   Iodine dosage is 5-10 drops daily of Lugol's solution or
   saturated solution of potassium iodide. In mild thyrotoxi-

cosis, iodine therapy alone for 10-15 days before operation may be sufficient preparation.

Complications of Hyperthyroidism.

A. Acute Hyperthyroid Crisis (Thyroid Storm): This is usually precipitated by surgery in a patient with severe thyrotoxicosis. Since the advent of antithyroid drugs it is possible to control hyperthyroidism before surgery in most instances. The major features include restlessness, delirium, tachycardia, hyperpyrexia, and vasomotor collapse. The prognosis is grave (mortality over 50%). An "apathetic" type also occurs which is characterized by hypotonia, apathy, and prostration with only mild pyrexia. Treatment is urgent and consists of controlling hyperpyrexia with cold packs and restlessness with sedation. Sodium Iodide, U.S.P., 1-2 Gm. (15-30 gr.) I.V. every 12-24 hours, and large doses of an intravenous corticosteroid (see p. 607) are advocated.

B. Severe Exophthalmos: Exophthalmos is the result of edema and later cellular infiltration of the orbital tissues, theoretically caused by anterior pituitary hormone. The probable inhibitory effect of thyroid secretion on the anterior pituitary gland is the reason for giving thyroid hormone in exophthalmos. Removal of the thyroid does not necessarily relieve exophthalmos, and may even aggravate it. In marked exophthalmos there is danger of corneal ulceration, severe limitation of extraocular muscle movement, and blindness. Significant or progressive exophthalmos is an indication for periodic examination with an exophthalmometer. The following treatment measures should be considered:

1. Protection of the eyes with dark glasses during the day and eye shields at night. Ophthalmologic consultation should be obtained. In extreme cases, tarsorrhaphy may be necessary.
2. Thyroid hormone is begun when the BMR reaches +20% during propylthiouracil or other therapy or when exophthalmos tends to progress after thyroidectomy. Begin with 0.1-0.2 Gm. daily and adjust dosage to maintain the BMR at about +20%. This therapy is not invariably effective.
3. Corticosteroids or corticotropin (ACTH) in large doses is reported to be helpful (see p. 607) for certain patients.
4. Orbital decompression may rarely be necessary to save the vision if exophthalmos progresses in spite of conservative therapy.

Prognosis.

The results of subtotal thyroidectomy for toxic nodular goiter are generally good, and the incidence of recurrent thyrotoxicosis or hypothyroidism is quite low. The recurrence rate following thyroidectomy for Graves' disease is about 10%. Hypothyroidism occurs in about 10% of cases. Operative mortality in experienced hands is as low as 0.25%.

SOLITARY THYROID NODULE

Ten to 20% of solitary thyroid nodules are malignant; the re-mainder consist chiefly of adenomas and nodular goiter in approxi-mately equal proportion. Unfortunately, there is no certain means of distinguishing benign from malignant nodules. Both are usually asymptomatic incidental findings. A helpful diagnostic procedure is to measure the thyroid region with a scanning device after ad-ministration of a test dose of radioactive iodine. If the nodule takes up radioactive iodine it is probably neither adenoma nor cancer. "Cold" nodules must be looked upon with more suspicion.

Single thyroid nodules occur more frequently in women than in men. Neither youth nor long duration rule out cancer, since thy-roid malignancy (particularly papillary carcinoma) appears even in the teens and may be present for months or years without dis-cernible change in size. These lesions should therefore be excised completely, submitted immediately for frozen section, and the surgical procedure planned accordingly.

THYROID ADENOMA

Thyroid adenomas are benign, encapsulated, slowly-growing neoplasms which are usually single but may be multiple. Clinically they present as firm, nontender, discrete, ovoid nodules. Ade-nomas rarely become malignant, but they must be removed because they cannot be differentiated from cancer except on microscopic examination of a frozen section in the operating theater. The com-monest cell type is the follicular adenoma, which may be difficult to distinguish pathologically from follicular carcinoma since blood vessel invasion and hematogenous metastases can occur in benign-appearing lesions.

THYROID CARCINOMA

There are 3 general types of thyroid carcinoma: papillary (60%), follicular (25%), and undifferentiated (15%). The nodule is firm and nontender, and there are wide variations in rate of growth. Pressure symptoms and recurrent nerve palsy are characteristic of undifferentiated lesions.

Papillary Thyroid Carcinoma.

All ages may be affected, but most patients are under 40. Papillary carcinoma occasionally grows very slowly over a period of 20-30 years, but the assumption that papillary carcinoma can be treated as a benign neoplasm is not warranted. Actually, this is an unpredictable tumor which invades the regional nodes in about 75% of cases and involves the opposite lobe of the gland in about 30%. Treatment is controversial, but should probably consist of total thyroidectomy with simultaneous ipsilateral complete neck dissection if the nodes are clinically suspicious or positive on frozen section. Complete neck dissection on the side of the tumor is advocated by some authorities even in the absence of demon-strable nodes. Metastases can sometimes be temporarily inhibited

with Thyroid, U.S.P., 0.2 Gm. (3 gr.) daily. The five-year clinical cure rate in papillary carcinoma is about 80%.

Follicular Thyroid Carcinoma.

This type of thyroid cancer usually occurs in patients over 40 years of age. It tends to spread via the blood stream, especially to the bones and lungs, but lymphatic metastases and spread to the opposite lobe are also common. Surgical treatment is the same as for papillary carcinoma. Metastases of follicular cancer may be sufficiently biologically active to permit their localization and palliative treatment with radioactive iodine. The five-year clinical cure rate in follicular carcinoma is about 60%.

Undifferentiated Thyroid Carcinoma.

These lesions may be of the small cell, giant cell, or epidermoid variety. They occur after the age of 40 and spread rapidly. Surgical extirpation is often impossible. These tumors do not take up radioiodine, and palliative radiotherapy is the only recourse in nonresectable cases. The five-year survival rate is about 25%.

THYROIDITIS

The various types of thyroiditis are of surgical importance chiefly because they may be confused with thyroid cancer.

Subacute thyroiditis is an inflammatory process, possibly viral in origin, which causes tender enlargement of 1 or both lobes often accompanied by moderate fever, pain on swallowing, and pain radiating to the ears. Women are affected 6 times as often as men, and the average age at onset is 45 years. The course of the illness is about 10 days. Palliative effects can be obtained with Cortisone Acetate, U.S.P., 25 mg. q.i.d. orally, or x-ray therapy.

Struma lymphomatosa (Hashimoto's struma) is a disease exclusively of females (average age about 45) characterized by chronic, firm, diffuse, nontender, bilateral enlargement of the thyroid gland. The etiology is uncertain, but an autoimmune reaction may be responsible. Hypothyroidism usually develops. In rare cases pressure symptoms may require excision of the isthmus and portions of the lobes. Thyroid therapy is usually indicated.

Riedel's struma is a rare form of thyroiditis in which the gland is fibrotic, hard, adherent, and slightly enlarged, often on 1 side only. Women are affected twice as often as men, and the average age at onset is about 45. The etiology is not known. Pressure symptoms are the only indication for surgery, but biopsy may be necessary to rule out carcinoma. Myxedema eventually occurs and calls for treatment with thyroid hormone.

HYPERPARATHYROIDISM

Excessive secretion of parathyroid hormone may be caused by parathyroid adenoma (90%), hyperplasia (10%), or carcinoma (rarely). The result is an increased loss of calcium and phosphorus from the body. Elevated calcium frequently leads to the formation of renal calculi, the commonest complication of hyperparathyroidism.

When oral intake of calcium is insufficient to compensate for urinary loss, the bones become decalcified.

Clinical Findings.
  A. Symptoms and Signs: Lassitude and fatigability are the earliest symptoms, but are so nonspecific that they rarely suggest the diagnosis. Polydipsia and polyuria may also occur. Hyperparathyroidism is rarely suspected until renal colic or bone pain develops. It is important to consider hyperparathyroidism in all cases of kidney stones. Peptic ulcer and acute pancreatitis may occur with increased frequency in patients with hyperparathyroidism.
  B. Laboratory Findings: Serum calcium is elevated, serum phosphorus is low, and urinary excretion of calcium is increased (positive Sulkowitch test). Alkaline phosphatase is increased if bone disease is present; a normal level of alkaline phosphatase rules out skeletal involvement.
  C. X-ray Findings: Decalcification, pathologic fracture, or osteitis fibrosa cystica is found in 10-30% of patients. The most important sites to study for bone abnormality are the hands, teeth, and skull. Subperiosteal reabsorption of bone is pathognomonic. Absence of the lamina dura around the teeth is highly suggestive.

Complications.
    Renal failure is a common complication of hyperparathyroidism. A significant number of patients die of uremia after successful surgical relief of the hyperparathyroidism. This emphasizes the importance of early diagnosis.

Treatment.
    Once the diagnosis is established on chemical grounds, the only satisfactory treatment is surgical. The thyroid area is explored until all of the parathyroids are located. This may also entail exposure of the retrotracheal and retro-esophageal areas and the anterior and posterior mediastinum. All adenomas should be excised. If there is hyperplasia of all 4 parathyroids, 3 should be removed and the fourth subtotally resected. In over 80% of patients there are 4 parathyroid glands, but in about 6% there are 5 and in rare cases only 3 are present. Adenomas are usually single, but in 10% there are 2 and in rare cases 3. In about 1% an adenoma is found in the anterior mediastinum. Hyperparathyroidism disappears rapidly after proper treatment.
    Transient hypocalcemic tetany occurs in about 50% of cases, usually 2-4 days after surgery. When generalized decalcification of the skeleton is present and when the serum alkaline phosphatase level is elevated to 10-20 Bodansky units, rapid absorption of calcium by the bone with tetany can be anticipated postoperatively. Treatment is as outlined on p. 194. Several months may be required for the remaining glands to produce sufficient hormone to abolish symptoms.

COMPLICATIONS OF THYROIDECTOMY

HYPOTHYROIDISM

Postoperative myxedema occurs in about 10% of patients in whom a radical subtotal thyroidectomy is done for Graves' disease. The complication is less common after removal of toxic nodular and other forms of goiter.

Symptoms and signs include weakness, fatigability, intolerance to cold, dry skin, and falling hair (especially the eyebrows), and the face becomes puffy. The BMR is −30% or below, and radioactive iodine uptake is low (about 10% in 24 hours). Blood cholesterol rises.

Administer Thyroid, U.S.P., 30 mg. ($1/2$ gr.) daily, increasing by 30 mg. ($1/2$ gr.) every week until hypothyroidism is controlled. Maintenance dosage is usually 60-130 mg. (1-2 gr.) daily as judged by clinical response and protein-bound iodine or BMR.

In severe myxedema and in the presence of heart disease, begin with small doses, 8-15 mg. ($1/8$-$1/4$ gr.) daily, and increase by 15 mg. ($1/4$ gr.) each week until optimum effect is obtained.

Sodium Liothyronine, N.N.D. (Cytomel®), is a rapidly absorbed synthetic drug which produces thyroid effects within 24-48 hours. When a rapid response is desired, it may be used instead of thyroid. Begin with 5 mcg. daily and increase by this amount weekly. Cytomel®, 25 mcg., is equivalent to thyroid, 60 mg. (1 gr.).

HYPOPARATHYROIDISM

Damage to or removal of the parathyroid glands resulting in transient or permanent hypoparathyroidism occurs in about 3% of thyroidectomies. Temporary deficiency of parathyroid hormone (for several months) may develop after excision of a parathyroid adenoma.

Clinical Findings.
Muscle weakness, irritability, numbness or cramps in the extremities, tetany, carpopedal spasm, laryngeal stridor, and Chvostek's sign are among the characteristic features. Frequently the only clinical evidence of impending tetany is Chvostek's sign. The diagnosis is confirmed by the finding of a low serum calcium and a high or normal serum phosphorus. Symptoms may occur within 2-7 days following surgery, or the disorder may remain latent, causing only vague complaints. Cataracts, convulsive disorders, and mental deterioration are late complications of chronic deficiency.

Treatment.
A. Emergency Treatment of Postoperative Tetany: Give immediately 1 of the following until tetany subsides: (1) Calcium Gluconate Injection, U.S.P., 10-30 ml. of 10% solution slowly I.V.; or (2) Calcium Chloride, U.S.P., 5-10 ml. of 10% solution slowly I.V. Ten to 50 ml. of either solution may be added to 1000 ml. of 5% glucose in water or saline for continuous administration if necessary.

B. Maintenance Therapy:
   1. Calcium salts are administered orally as soon as possible.
      Give calcium lactate or gluconate, 3-4 Gm. (45-60 gr.)
      q.i.d.
   2. High-calcium, low-phosphorus diet (omit milk).
   3. Calciferol, U.S.P. (Vitamin D_2), 50,000 units daily; may
      increase to 50,000 units q.i.d. if necessary.
   4. Aluminum hydroxide with magnesium hydroxide or magnes-
      ium trisilicate, 15 ml. (or two 0.5 Gm. tablets) q.i.d., is
      given by mouth to reduce absorption of phosphorus from the
      intestinal tract.
   5. If the above measures fail to maintain the blood calcium at
      a normal level, add Dihydrotachysterol, N.N.D. (Hytakerol®),
      0.5 ml. t.i.d. Both dihydrotachysterol and calciferol pro-
      mote calcium absorption from the intestine and decrease
      phosphorus resorption from the kidney tubules. Dihydro-
      tachysterol is more effective but is quite expensive.
   6. Regulation - The patient can be taught to regulate his own
      dosage of calciferol or dihydrotachysterol on the basis of a
      self-administered daily Sulkowitch test, which should be
      1-2+ on the early morning urine. Continuing long-term
      follow-up is essential to prevent serious sequelae. Periodic
      determination of blood calcium, phosphorus, and proteins is
      important in management.

RESPIRATORY OBSTRUCTION

Wound hemorrhage, laryngeal edema, tracheal collapse, or
bilateral cord palsy may cause airway obstruction in the immediate
postoperative period. All thyroidectomy patients should be ob-
served closely for such danger signals as stridor and dyspnea, since
severe hypoxia and death may occur shortly after the onset of these
symptoms. If significant respiratory distress develops, the wound
must be opened immediately and tracheostomy performed (see p.
166). It is preferable to take the patient to the operating room for
this procedure, but it may be necessary to do an emergency life-
saving tracheostomy on the ward. The postoperative orders on
every thyroidectomy patient should specify that a tracheostomy set
be kept at the patient's bedside.

RECURRENT NERVE PALSY

Operative injury of 1 or both recurrent nerves occurs in about
1% of thyroidectomies. Depending upon the degree of damage, cord
palsy may be temporary or permanent. If 1 nerve is injured,
hoarseness is apparent immediately after operation and persists for
several months. Hoarseness gradually decreases as the nerve re-
covers or as laryngeal function compensates for the paralyzed cord.

No treatment is of value. Bilateral cord palsy is a serious com-
plication usually leading to respiratory obstruction and necessitating
tracheostomy immediately after operation. It is good practice to ob-
serve cord function preoperatively by indirect laryngoscopy and to re-
peat the examination after surgery if hoarseness or stridor develops.

9 . . .

Surgery of the Pulmonary System

PULMONARY FUNCTION STUDIES

Normal lung function depends upon (1) pulmonary ventilation, (2) diffusion of gases across the alveolar-capillary membrane, and (3) pulmonary blood flow. The simplest and most widely available pulmonary function tests are measurements of ventilation. Specialized studies of diffusion and blood flow are less frequently required for diagnostic purposes.

Pulmonary function tests are of value in assessing operative risk, in the differential diagnosis of pulmonary disease, and as a means of determining response to treatment. In the great majority of cases a satisfactory preoperative estimate of lung function can be made on the basis of history, physical examination, chest x-ray, and fluoroscopy. In borderline cases, function studies provide objective evidence of pulmonary reserve. For detailed information on pulmonary physiology the reader is referred to Comroe & others: The Lung; Clinical Physiology and Pulmonary Function Tests. (Year Book, 1955.)

Bronchospirometry consists of inserting a double lumen catheter into the trachea and 1 of the main bronchi so that separate measurement can be made of tidal volume, minute volume, vital capacity, and oxygen consumption in each lung. This is of value in the rare instance when it is desirable to learn whether a patient facing pulmonary surgery will retain adequate pulmonary function after the operation.

DIAGNOSTIC AND THERAPEUTIC PROCEDURES OF VALUE IN THORACIC SURGERY

General Measures in Thoracic Surgical Care.
The immediate objective of management after thoracic surgery or trauma is complete expansion of the lung without residual air or fluid in the chest. The following measures serve this end:
A. Position and Ambulation:
   1. The patient should be placed in a semi-sitting position as soon as tolerated; this makes breathing and coughing more efficient.
   2. Pneumonectomy patients should lie alternately on the back and on the operated side; if such a patient is placed on his normal side severe respiratory embarrassment may result.
   3. Ambulation should be started as soon as possible (usually on the day following operation). Breathing exercises and other exercises to mobilize the shoulder girdle and thoracic cage should be encouraged throughout the convalescent period.

B. **Prevention of Atelectasis:**
   1. Deep breathing and repeated vigorous cough at least hourly are essential in the early postoperative period.
   2. Tracheal aspiration is employed as necessary (see p. 53). Bronchoscopy is occasionally useful.
   3. Positive-pressure breathing with a bronchodilator or wetting agent should be used both preoperatively and postoperatively if secretions are marked or bronchospasm is present (see p. 54).
   4. Expectorants may be of value (see p. 610).
C. **Pain Relief:** The dosage of narcotics given to thoracic patients should be strictly individualized. Pain relief sufficient to make coughing bearable is essential, but depression of respirations and cough reflex must be minimized. Barbiturates can often be used for sedation to reduce the need for narcotics. Blocking of the intercostal nerves with procaine from several spaces above to several spaces below the level of the operative wound or site of chest injury may occasionally be of value.
D. **Oxygen Therapy:** Pulmonary insufficiency is often present during the first 24-48 hours after chest surgery or severe trauma. Oxygen by nasal catheter (at 5-7 L./minute) is usually given routinely after thoracotomy until pulmonary function is adequate. Dyspnea and cyanosis are indications for resumption of oxygen therapy. When pulmonary reserve is markedly diminished, tracheostomy may be lifesaving by reducing respiratory dead space and permitting adequate removal of secretions.
E. **Tracheostomy:** Markedly diminished pulmonary reserve or excessive secretions which cannot be adequately removed by tracheal aspiration may be an indication for tracheostomy (see p. 166). Positive-pressure breathing through a tracheostomy may be life-saving and should be instituted promptly when oxygenation is inadequate in spite of other measures.
F. **Management of Chest Drainage Tubes:** One or more chest tubes for postoperative drainage of fluid and air are usually placed at thoracotomy through the intercostal spaces. (Tubes are rarely used when a pneumonectomy has been performed.) Continuous functioning of these tubes is essential to ensure full lung expansion and obliteration of the pleural space. The tubes must be inspected every few hours, must not be blocked for long periods, and must never be opened to the air. Tubes should be attached at the operating table or immediately postoperatively to waterseal or gentle suction (see p. 201). When there is no pulmonary air leak, waterseal drainage is usually adequate. When an air leak from the lung is present, sufficient suction should be applied to the chest catheter so that bubbles appear in the waterseal bottle on expiration or cough but not on inspiration. The amount of suction usually required is 8-25 cm. of water. Strong suction (up to 40 cm. of water or more) to "pull out" the lung to the chest wall and seal the leak may be successful but occasionally causes such complications as excessive bleeding from denuded lung surfaces and dyspnea due to the aspiration of air from the tracheobronchial tree. Chest tubes are removed when they have been blocked for 24 hours.

   Alveolar air leaks may continue for days and occasionally weeks after segmental resection or lobectomy. These usually

MEASUREMENTS OF PRINCIPAL LUNG VOLUMES AND FORMULAS FOR THEIR PREDICTION IN NORMAL SUBJECTS*

	AGE 16-34		AGE 35-49		AGE 50-69	
	Women	Men	Women	Men	Women	Men
Vital capacity, supine (ml.)	2312-4150	2792-4950	2212-3435	3300-5240	1570-3525	2184-5429
Maximum breathing capacity, standing (L./min.)	63.6-117.5	82-169	47-114	86-144.5	49-101.5	58-139
Ventilation, resting (L./min./sq. M. body surface)	2.55-4.27	3.1-4.5	2.4-3.71	2.6-4.0	2.53-3.95	3.2-4.9
Oxygen consumption, resting (ml./min./sq. M. body surface)	111-149	129-186	109-136	118-156	105-150	107-165
Predicted (calculated) total capacity (supine)	$\dfrac{\text{Vital Capacity}}{80} \times 100$		$\dfrac{\text{Vital Capacity}}{76.6} \times 100$		$\dfrac{\text{Vital Capacity}}{69.2} \times 100$	
Ratio $\dfrac{\text{Residual Volume}}{\text{Total Capacity}} \times 100$ (supine)	20		23.4		30.8	
Predicted (calculated) vital capacity, supine (ml.)	Women: [21.78 − (0.101 × age in yrs.)] × height in cm. Men: [27.63 − (0.112 × age in yrs.)] × height in cm.					
Predicted (calculated) maximum breathing capacity, standing (L./min.)	Women: [71.3 − (0.474 × age in yrs.)] × sq. M. body surface Men: [86.5 − (0.522 × age in yrs.)] × sq. M. body surface					

*From E. de F. Baldwin, A. Cournand, and D. W. Richards, Jr.: Medicine, Vol. 27. Reproduced with permission.

Normal Values of Arterial Gas Analysis at Rest:
1. Oxygen Saturation: 96-99%
2. CO_2 Content: 25-28 mM./L. (plasma)
3. CO_2 Tension: 41 ± 3 mm. Hg
4. pH: 7.42 ± 0.02 at 37°C.

seal if tube drainage and suction are maintained. If an undrained air space of significant size persists, a tube should be placed in it for closed drainage. Small, uninfected air pockets usually absorb completely without drainage.

Bronchopleural fistula (an air leak from a bronchus into the pleural space) may or may not be associated with an empyema. The fistula usually opens within the first 1-2 weeks after pulmonary resection. Many will close on prolonged tube drainage; others require open drainage by rib resection. When simpler measures fail, suture of the fistula or thoracoplasty is necessary. Thoracoplasty is usually required for chronic fistula after pneumonectomy or in tuberculosis

G. Chest Fluid and Empyema· Fluid or blood occasionally accumulates in the chest in spite of tube drainage. Small amounts of uninfected fluid and blood will absorb without incident. Fluid of sufficient volume to be apparent on x-ray should be removed as completely as possible by thoracentesis and cultured. If the fluid is infected (empyema), complete evacuation by repeated thoracenteses or by trocar thoracotomy (see p. 201) is indicated. Until sensitivity tests can be completed, 1 million units of penicillin and 0.5 Gm. of streptomycin should be placed in the chest cavity at the conclusion of each thoracentesis. Streptomycin and penicillin should be given systemically also. The antibiotic regimen can be altered as necessary when sensitivity studies are complete.

Complete removal of air and fluid from the chest and full expansion of the lung postoperatively are the most important means of preventing empyema.

Thoracentesis.

A. Preparation of the Patient: The site of the tap is selected by x-ray, fluoroscopy, or physical examination. When there is a large basilar effusion or total hydrothorax, the best site of aspiration is usually at the angle of the scapula or in the seventh or eighth interspace in the posterior axillary line. The patient is placed in a comfortable sitting position, leaning slightly forward on a padded stand or supported by an attendant. Premedication with a barbiturate or narcotic is optional. The skin of the chest wall is prepared with antiseptic and draped.

B. Technic of the Tap: (Aseptic technic must be used throughout.) The entire thickness of the chest wall, including the parietal pleura, is infiltrated with 5-10 ml. of 1% procaine through a No. 22 gauge needle. Thoracentesis is virtually painless if this is properly done. A short-bevel needle (7.5-10 cm. long, 18-13 gauge) is attached by a three-way stopcock to a 20-50 ml. syringe. With firm, steady pressure the needle is advanced into the pleural space. In order to avoid injury to the intercostal nerve and vessels, pass the needle through the chest wall at the lower margin of the intercostal space (except when making a tap on the anterior chest wall, in which case the needle is passed through the center of the interspace). A clamp may be placed on the needle to steady it at the chest wall. Care is taken during aspiration to prevent air from entering the chest.
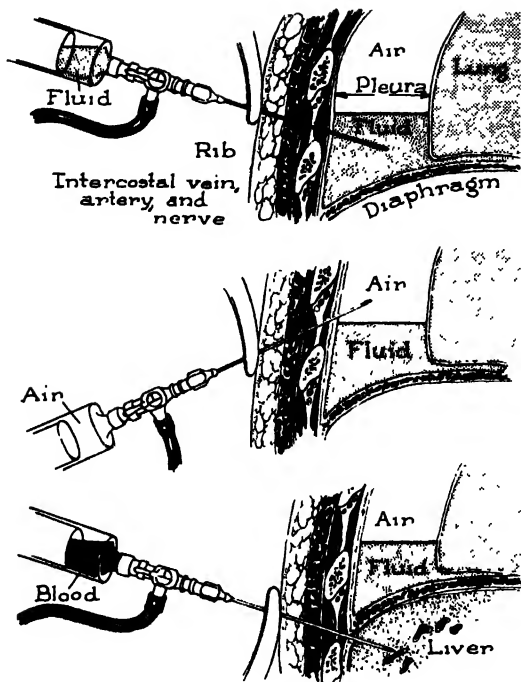
Thoracentesis. Top: A successful tap with fluid obtained. Note the position of the needle with relation to the intercostal bundle, and the use of the clamp to steady the needle at skin level. Center: Air is obtained as the needle is shifted upward. Bottom: A bloody tap results from an excessively low position of the needle with puncture of the liver. (Redrawn and reproduced, with permission, from G. E. Lindskog and A. E. Liebow, Thoracic Surgery and Related Pathology. Appleton-Century-Crofts, 1953.)

C. Volume of Fluid Removed: The amount of fluid removed at one sitting depends upon circumstances. A total of 2000 ml. can often be gradually evacuated at 1 sitting from a large effusion. If the patient complains of a feeling of tightness in the chest, cough, palpitation, fatigue, faintness, or other untoward symptoms, slow the rate of aspiration or discontinue until another occasion. If special radiographic studies or thoracoscopy will be of value, a small amount of air (50-100 cc.) is injected into the chest before removing the needle.

D. **Laboratory Examination of Pleural Fluid:** Laboratory exam-
ination of the fluid may include 1 or more of the following:
Total volume, odor, color, turbidity, viscosity, coagulability,
sp. gr.; smear for Gram's and acid-fast stains; total and dif-
ferential blood cell count; aerobic, anaerobic, and acid-fast
cultures; guinea pig inoculation; cytologic study for neoplastic
cells; histologic study of sediment from a centrifuged speci-
men.

Trocar Thoracotomy.

Closed drainage of the chest cavity with a catheter is required
when thoracentesis is unsatisfactory because of pulmonary air leak
or marked viscosity of the pleural fluid. The objective is to place
a catheter in the dependent portion of the fluid space when the pa-
tient is sitting. If possible, the posterior or midaxillary line is
selected (so that the patient will not lie on the tube). An upper an-
terior interspace is usually chosen for treatment of pneumothorax.
The procedure may be done in bed in a critically ill patient. Cath-
eter drainage is usually inadequate when chronic infection has re-
sulted in marked pleural thickening. Under these circumstances,
rib resection and open tube drainage of the empyema may be indi-
cated.
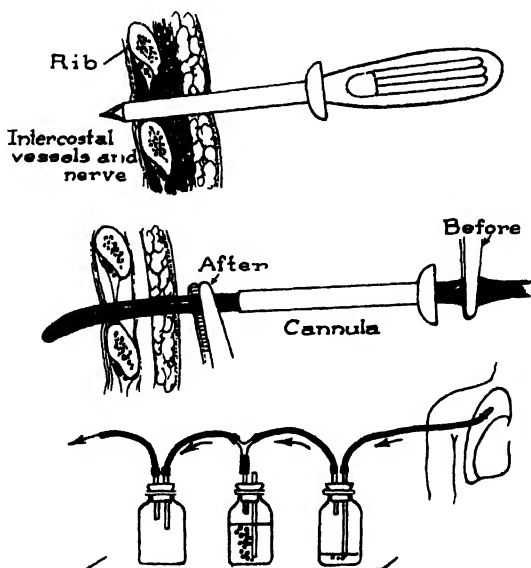
Trocar Thoracotomy. Top: Penetration of trocar into chest.
Center: Insertion of flexible catheter through cannula, and
removal of cannula. Bottom: Suction bottles with waterseal.

Preparation is as for thoracentesis The chest is tapped with a needle to establish the position of the fluid pocket. After infiltration with 1% procaine, a 1 cm. skin incision is made. A trocar of suitable size is introduced through the chest wall with firm pressure and rotary motion. A flexible catheter of the largest size which will pass through the trocar is passed into the chest. Several extra drainage holes are usually cut in the side of that portion of the catheter which will lie in the chest. The sheath of the trocar is removed and the catheter is secured to the chest wall with a nonabsorbable suture. The catheter is kept clamped during manipulation in order to prevent pneumothorax and is attached immediately to waterseal or suction drainage.

CHEST TRAUMA

RIB FRACTURES

Direct violence is the usual cause of rib fractures, but spontaneous fractures, usually of the sixth to ninth ribs, occasionally occur as a result of strenuous coughing. Pathologic fracture in a rib affected by metastatic malignancy or other bone disease is rare.

Pain, sharply localized to the fracture site and aggravated by breathing and other motions of the rib cage, is typical. Limitation of respiratory motion on the injured side is often present. Tenderness and sometimes crepitation are present at the fracture site. Severe injury may be complicated by subcutaneous emphysema, pneumothorax, and hemothorax Atelectasis and pneumonitis frequently follow rib fracture in elderly persons.

Fractured ribs over the spleen should always raise the question of associated rupture of that organ. Suspicion of rib fracture is always an indication for chest x-ray. Special views may be required to show the involved region. Fracture separations at the costochondral junctions do not visualize radiologically.

The pain of uncomplicated rib fracture may be diminished by strapping the chest with adhesive after shaving the skin and applying a coating of tincture of benzoin or other adhesive agent. Strapping should extend from the anterior axillary line on the sound side, around the involved hemithorax to the posterior axillary line on the injured side. Each strip of adhesive is applied with the patient holding his breath in forced expiration. Multiple fractures with hypermobility of a segment of chest wall are an indication for strapping to control paradoxical motion.

Intercostal nerve block, performed as soon after injury as possible, is very useful in severe multiple fractures accompanied by respiratory distress. Five to 10 ml. of 1% procaine are injected paravertebrally just beneath the lower margins of the involved ribs and several normal ribs above and below this level. The injection may be repeated as often as necessary.

Rib fractures may lead to atelectasis and serious pulmonary infection in elderly patients. Tracheobronchial secretions in these patients must be removed as often as necessary by coughing and by tracheal aspiration if necessary. Antibiotic therapy is frequently

required to control pneumonitis. Depression of cough and respiration by excessive doses of narcotics should be avoided. Strapping of the chest for uncomplicated rib fracture in elderly patients is usually contraindicated as predisposing to atelectasis.

FRACTURE OF THE STERNUM

Fracture of the sternum is caused by severe direct trauma, as in steering wheel injury. Multiple rib fractures and other injuries are usually also present. Contusion of the heart is not uncommonly present. Severe precordial pain and dyspnea are the chief symptoms. Crepitus and deformity of the sternum may be noted. In most cases the only treatment required is rest in the sitting position, pain relief with narcotics, and strapping of associated rib fractures. When there is significant deformity, either closed or open reduction under local anesthesia should be carried out. Hypermobility of the anterior chest wall secondary to multiple fractures can be stabilized by overhead traction. When severe respiratory difficulty cannot be controlled by other measures, positive-pressure breathing through a cuffed tracheostomy tube may be lifesaving. This can be continued for days or weeks until the chest wall is stabilized.

HEMOTHORAX

Bleeding from the lung parenchyma tends to be self-limited. Continued bleeding usually originates in the chest wall, diaphragm, mediastinum, or hilus. Several liters of blood may accumulate in the thorax. In three-fourths of patients the blood remains fluid. In the absence of persistent bleeding, hemothorax can be treated successfully in 80-90% of cases by multiple aspirations or trocar thoracotomy.

Symptoms and signs depend upon the extent of the hemothorax and the associated injuries. If large collections of blood are present, shock and physical signs of chest fluid are seen. Definitive diagnosis is made by x-ray.

Treatment.
A. Emergency Measures: Treatment in massive hemothorax is directed toward immediate replacement of blood loss (see p. 112), and thoracentesis to relieve respiratory distress by removing blood and air from the chest. Other general supportive measures are also instituted (see p. 196). If marked hemorrhage continues or recurs after the original thoracentesis (as indicated by reappearance of shock and increase in chest fluid) emergency thoracotomy is advisable. If symptoms are stabilized by the initial treatment, thoracentesis is repeated within the first 24-48 hours as often as necessary to evacuate all blood and permit complete lung expansion. When hemopneumothorax is present, the early placement of chest tubes by trocar thoracotomy through the upper anterior and lower posterolateral chest wall is the best means of expanding the lung promptly.

B. Management of Clotted Hemothorax: Rapid clotting or locula-
tion of hemothorax occasionally prevents satisfactory needle
aspiration. Under these circumstances, enzymatic debride-
ment or decortication should be considered. Enzymatic de-
bridement is accomplished by injecting into the hemothorax a
solution of fibrinolytic enzyme. Streptokinase-Streptodornase,
N.N.D. (Varidase®), is most widely used for this purpose. A
solution of 100,000-200,000 units of streptokinase and 25,000-
50,000 units of streptodornase dissolved in not less than 10 ml.
of saline is injected into the hemothorax, and the pleural space
is aspirated completely after 12-18 hours. This usually re-
sults in an outpouring of pleural fluid, and repeated thoracen-
teses may be necessary to remove it. Further injections of
the enzymes may be given as required. Malaise, chest pain,
and fever following the injection usually subside rapidly and
can be controlled with analgesics.

Decortication of the lung with evacuation of the residual
debris is reserved for the patient with a large organizing
hemothorax. The ideal time for surgical intervention is be-
tween the third and fifth week following injury, but other con-
siderations (e.g., if infection is present or if conservative
measures are obviously inadequate) may make earlier opera-
tion advisable.

TRAUMATIC PNEUMOTHORAX

Pneumothorax may be caused by an open "sucking" wound in
the chest wall or, more commonly, an air leak in an injured lung.

Sucking Wounds of the Thorax.

These must be closed immediately with a bulky occlusive
dressing held in place by adhesive or a bandage or binder encir-
cling the chest. Vaseline gauze over the wound assists in sealing
the leak. Open pneumothorax causes marked to and fro shifting of
the mediastinum during respiration ("mediastinal flutter") which
must be promptly controlled to prevent respiratory and circulatory
failure. Surgical closure of the wound in the chest wall is carried
out as soon as the patient's condition permits. Meanwhile, air is
evacuated from the chest as described below.

Closed Pneumothorax.

Closed pneumothorax is caused by an air leak from the lung or
tracheobronchial tree. It may occur either as a result of a pene-
trating chest wound or blunt injury.

A. Simple Pneumothorax: Pneumothorax accompanies many blunt
and penetrating chest injuries and is often associated with
hemothorax. When lung collapse is no more than about 10%,
special treatment of the pneumothorax is usually not necessary.
Early and complete expansion of the lung with removal of all
air and fluid from the chest is the primary objective of local
treatment when a significant pneumothorax is present. Occa-
sionally this can be accomplished by 1 or more thoracenteses
during the first 24-48 hours. If there is lung collapse of more
than 25% initially or if pneumothorax recurs after thoracen-
tesis, trocar thoracotomy with placement of a catheter in the

upper chest through the second or third interspace anteriorly is probably indicated. Gentle suction (15-25 cm. of water) is applied for 3-5 days or until blocking of the catheter indicates that expansion is complete and the lung adherent.

B. Tension Pneumothorax: See p. 221.

SUBCUTANEOUS AND MEDIASTINAL EMPHYSEMA

Rib fractures, chest wounds, traumatic rupture of the trachea or a major bronchus, spontaneous pneumothorax, and pulmonary resection are occasionally followed by subcutaneous emphysema. In advanced cases, emphysema extends from head to foot and produces an alarming degree of swelling and crepitation, especially of the head, neck, and scrotum. Air in the tissues causes little harm and, as a rule, requires no treatment.

Attention should be directed toward correction of the underlying cause. Pneumothorax should be controlled by trocar thoracotomy, and irritating, useless cough should be minimized. Major air leaks in the lung or tracheobronchial tree may require surgical repair.

Marked emphysema of the mediastinum will on rare occasions produce sufficient tension to threaten life. If dyspnea, cyanosis, tachycardia, and shock progress in spite of other measures, tracheostomy or cervical mediastinotomy is indicated. Mediastinotomy (under local anesthesia) consists of blunt dissection into the superior mediastinum through a transverse incision in the suprasternal notch to permit the egress of air.

TRAUMATIC RUPTURE
OF THE TRACHEA OR BRONCHI

Severe crushing or compression trauma to the chest may cause injury to the trachea or major bronchi. Bronchial rupture, usually within 1-2 cm. of the carina, occurs about 4 times as often as tracheal tear. Tracheobronchial injuries are often overlooked, as there may be no other apparent injury to the chest.

Symptoms and signs include dyspnea, subcutaneous emphysema, cyanosis, pain, hemoptysis, shock, cough, pneumothorax, extensive atelectasis, and hemothorax. The diagnosis may be overlooked until weeks or months after the injury. X-rays then usually show complete atelectasis of the involved portion of lung.

Treatment is by suture of the tear if the patient's other injuries do not contraindicate thoracotomy. Emergency operation may be required to relieve airway obstruction or to correct uncontrollable air leak, tracheobronchial hemorrhage, or rapidly advancing mediastinal emphysema. Chronic stricture with obstruction requires bronchoplastic operation. The distal lung, although collapsed, is often functional when reexpanded; pulmonary resection should therefore be reserved for cases with irreversible damage.

THORACOABDOMINAL WOUNDS

Left-sided wounds are more serious than those on the right because they are more frequently associated with injury to the spleen, stomach, and the splenic flexure; wounds on the right usually involve only the liver and are less likely to cause continuing hemorrhage or massive contamination of the chest and peritoneum. For this reason, suspected penetrating injury to the left diaphragm is an indication for early operation. The principal indication for intervention in right-sided involvement is continued hemorrhage.

The diaphragm may rise as high as the anterior fourth interspace on full expiration, and is thus vulnerable to penetration by injuries of the lower chest. The mechanism of the injury or the trajectory of a missile often indicates that thoracoabdominal injury has probably occurred. Additional information is obtained on physical examination and upright films of the chest and abdomen. Thoracentesis may disclose stomach or colon contents in the chest. The absence of signs of peritoneal irritation does not rule out damage to the abdominal organs.

In left thoracoabdominal injury, treatment is by thoracotomy as soon as the patient's condition permits. Right thoracoabdominal wounds require close observation and supportive measures; intervention is generally reserved for patients with continued bleeding or extensive visceral damage.

TRACHEOBRONCHIAL FOREIGN BODY

Infants, children, and intoxicated, anesthetized, and unconscious individuals are most likely to aspirate foreign bodies. Coughing, choking, and cyanosis may occur immediately after inhalation but often subside for a variable period. Depending upon the size, nature, and site of lodgement of the foreign body, later findings may include cough, wheezing, atelectasis, or pulmonary infection. Unless the foreign body is radiopaque, x-ray evidences are indirect: obstructive emphysema, atelectasis, or pneumonitis. Intermittent findings of this nature, especially in children, are highly suggestive of foreign body. Bronchoscopy and possibly bronchography should be performed. Bronchoscopic removal should be attempted. If successful, inflammatory reactions usually subside promptly; if unsuccessful, thoracotomy and bronchotomy or resection are necessary. Foreign bodies in the tracheobronchial tree should always be removed, since prolonged retention usually leads to bronchiectasis or abscess formation requiring pulmonary resection.

DISEASES OF THE CHEST WALL

PECTUS EXCAVATUM
(Funnel Breast)

Pectus excavatum is a congenital, hereditary malformation characterized by depression of the sternum below the gladiomanubrial

junction with symmetric inward bending of the costal cartilages. As the infant develops, kyphoscoliosis also occurs. Severe degrees of funnel breast may embarrass pulmonary and cardiac function, but as a rule the only disturbance is cosmetic. Operative treatment may be indicated for either reason. The best results are obtained between the ages of 4 and 8, although surgery cannot completely eradicate the cosmetic blemish.

CERVICAL RIB

Supernumerary rib in the cervical region, usually bilateral, is seldom of any clinical significance. When symptoms occur, they frequently involve 1 side only, and are caused by compression of the subclavian artery and a portion of the brachial plexus between the scalenus anticus muscle and the cervical rib. Numbness, tingling, and coldness of the hand and forearm on the involved side are the usual complaints. The most common physical finding is diminution or disappearance of the radial pulse when the patient inspires deeply with his neck extended and his head turned to the affected side (Adson's test). Other signs in the affected extremity may include coolness (occasionally cyanosis), diminished pulse and BP, and atrophic changes of the skin and nails. Vasoconstrictive episodes similar to Raynaud's disease may occur. Poststenotic dilatation of the subclavian artery with aneurysm formation is seen in severe cases. The cervical rib is readily identified by x-ray. Similar symptoms in the absence of a cervical rib constitute the scalenus anticus syndrome

Mild cases are treated by postural exercises and physical therapy to improve shoulder girdle tone. If these measures fail the scalenus anticus muscle can be divided surgically and the anterior portion of the cervical rib resected. Minor aneurysmal dilatations of the subclavian artery do not require resection if the proximal constriction is relieved

DISEASES OF THE PLEURA

ACUTE EMPYEMA

The microorganisms which most frequently cause empyema are the pneumococcus, streptococcus, staphylococcus, and tubercle bacillus. Infections with coliform bacilli, Proteus vulgaris, Friedländer's bacillus, various fungi, and mixed organisms are rare. Invasion of the pleural space occurs in 1 of the following ways: (1) direct spread from a pneumonic focus, (2) rupture of a pyogenic or tuberculous abscess, (3) contamination as a result of operation or trauma, (4) extension from below the diaphragm, and (5) hematogenous infection.

The principles of treatment in empyema are early evacuation of all fluid and purulent exudate in order to achieve complete lung expansion, and eradication of the infecting organism by adequate drainage, antibiotic therapy, and supportive measures.

Clinical Findings.

The diagnosis of acute empyema is seldom difficult. A predisposing condition is usually apparent. Findings related primarily to the empyema usually consist of chest pain, cough, malaise, fever, and leukocytosis. Large acute effusions may be associated with considerable toxicity and dyspnea. Physical examination discloses signs of pleural fluid; more accurate localization is provided by chest films. If air is seen in the empyema cavity on x-ray, it can be assumed that a bronchopleural fistula has developed. Thoracentesis (see p. 199) is done promptly in order to obtain material for smears, cultures, and antibiotic sensitivity studies. Fifty to 100 cc. of air may be injected into the cavity to facilitate x-ray study.

Treatment.

A. Antibiotic Therapy: Systemic antibiotic therapy is usually indicated.

B. Thoracentesis: The empyema cavity is evacuated completely by thoracentesis each day until less than 50 ml. can be aspirated on successive days. If the infectious agent is sensitive to a suitable topical antibiotic, intrapleural injections can be given at the conclusion of each aspiration.

C. Enzymatic Debridement: If the empyema fluid becomes too thick for satisfactory removal through the needle, Streptokinase-Streptodornase, N.N.D. (Varidase®), may be injected intrapleurally in an attempt to thin the exudate (see p. 204).

D. Trocar Thoracotomy: If the exudate is too thick for complete removal by thoracentesis, a catheter should be introduced into the cavity by trocar thoracotomy in order to establish closed waterseal drainage (see p. 201). Intrapleural enzymatic debridement may make this mode of drainage more efficient. Trocar thoracotomy is usually advisable at the outset of treatment if a bronchopleural fistula is present as indicated by pyopneumothorax.

Only closed methods of pleural drainage (e.g., thoracentesis and trocar thoracotomy) should be used until the lung is firmly adherent to the chest wall around the empyema cavity. If open drainage is established before this occurs, total collapse of the lung may result.

Prognosis.

Postpneumonic empyema usually subsides promptly when treatment is begun early. Empyema of other etiology is often more complicated and the prognosis less satisfactory. When closed methods of management fail, treatment must be along the lines described below for chronic empyema.

CHRONIC EMPYEMA

As an empyema becomes chronic, the pleura thickens and adheres firmly around the encapsulated exudate. There may be a history of recent acute empyema, or chronic empyema may exist for years in a latent or intermittently symptomatic state.

Empyemas become chronic for the following reasons: (1) delay in diagnosis or inadequate treatment of the acute stage; (2) broncho-

pleural fistula; (3) specific infection, such as tuberculosis or actinomycosis; (4) retained foreign body, (5) osteomyelitis of a rib, (6) disease of the lung preventing expansion, and (7) underlying malignant neoplasm.

Clinical Findings.

Cough and recurrent fever are usually the principal complaints. When some form of suppurative lung disease is associated with the empyema, the cough is usually productive of frankly purulent and sometimes foul-smelling sputum. Chest pain, clubbing of the fingers, chronic malaise, dyspnea, anorexia, and weight loss may occur. Draining sinuses are occasionally present.

Physical examination discloses signs of chest fluid or thickened pleura. Respiratory excursions are usually limited on the affected side, and there may be contraction of the thoracic cage. These changes are usually readily apparent on x-ray.

Thoracentesis is performed to obtain material for examination of smears and cultures for pyogenic and acid-fast organisms.

Treatment.
  A. General Measures: Anemia, malnutrition, and debility due to chronic sepsis should be corrected. Systemic antibiotic therapy may be indicated as an adjunct to drainage or other local treatment.
  B. Specific Measures:
    1. Drainage - It is usually impossible to evacuate a chronic empyema cavity adequately by needle aspiration. If the exudate is relatively thin and the pleura not markedly thickened, closed drainage by trocar thoracotomy may be effective (see p. 201).
        Chronic empyema frequently requires open drainage. This is accomplished by resection under local anesthesia of a small segment of rib over the lower portion of the cavity. A large tube is inserted and is shortened gradually as the cavity slowly heals, which may require many weeks or even months. Irrigations are unnecessary if adequate drainage is maintained. The rate of healing may be determined by filling the cavity at weekly intervals with saline.
    2. Decortication - Patients in whom infection has been controlled by chemotherapy or drainage may be suitable candidates for decortication, in which thickened pleura is excised in order to permit full expansion of the lung. Expansion is carefully maintained postoperatively by closed tube drainage. Decortication combined with pulmonary resection is the treatment of choice in selected cases of bronchopleural fistula, bronchiectasis, lung abscess, and other disorders in which the underlying lung is severely damaged.
  C. Tuberculous Empyema: The management of tuberculous empyema, much less common since the advent of the antituberculosis drugs, is always complex. Bronchopleural fistula and secondary infection are frequently present. The proper timing and selection of therapeutic procedures requires experience and judgment. Consideration must be given to the systemic reaction of the patient, the extent of the tuberculosis, and the response to specific chemotherapy.

Tuberculous empyema (without secondary infection) may respond to general supportive measures, antituberculosis chemotherapy, and closed drainage by repeated thoracentesis or trocar thoracotomy. The trocar thoracotomy is usually placed in the anterior axillary line to reserve the posterolateral chest wall in case thoracoplasty or decortication should become necessary. Closed drainage is always used in order to avoid secondary pyogenic infection.

Mixed infections of the pleura, usually associated with bronchopleural fistula, may occasionally respond to intensive treatment along the same lines combined with penicillin or other antibiotic agent   In these cases, however, thoracoplasty or decortication, with or without resection, is usually required

NEOPLASMS OF THE PLEURA

Primary tumors of the pleura are very rare and are difficult to distinguish from malignancies arising in the lung, diaphragm, or elsewhere   The most frequent pleural neoplasm is the mesothelioma, which tends to spread diffusely in the pleural space. It occurs most commonly in men between 40 and 50   The chief complaint is usually pleural pain. Additional symptoms may include malaise, weakness, cough, dyspnea, weight loss, and fever. The physical and radiologic signs are those of pleural fluid and thickening. Fluid tends to reaccumulate rapidly after thoracenteses   The diagnosis is established by cytologic study of the pleural fluid or by pleural biopsy. Although pleuropneumonectomy has been carried out in some cases, no treatment is curative   Radiotherapy may have a transient palliative effect.

DISEASES OF THE LUNG

LUNG ABSCESS

The causes of pyogenic lung abscess are (1) necrotizing pneumonia, (2) aspiration of infected material or a foreign body, (3) septic embolus or infection of a pulmonary infarct, (4) bronchial obstruction by tumor, (5) infection of a cyst or bulla, (6) extension of bronchiectasis into the parenchyma, (7) penetrating chest wounds, and (8) transdiaphragmatic extension of infection, e.g., from a subphrenic or amebic abscess. When a lung abscess develops in childhood, suspect a foreign body. In the older age group, consider the possibility of bronchial obstruction by cancer

Clinical Findings.
  A. Symptoms and Signs: A history of a predisposing disorder is usually present. There may be a latent period of several days or weeks during which only malaise and fever are noted. Cough, pleurisy, chills, and fever occur as the process develops. Within a few days the patient may suddenly cough up a large amount of very foul, purulent sputum, usually blood-streaked

or frankly bloody. Copious, malodorous sputum associated with the debility of long-standing infection is typical of chronic abscess.

Signs vary with the size, position, and contents of the cavity and with the local pulmonary reaction. Clubbing of the fingers sometimes develops rapidly.

B. Laboratory Findings: Leukocytosis and anemia are usually consistent with severe infection. Sputum shows infection and may disclose the etiology, e.g., tuberculosis or neoplasm

C. X-ray Findings: Early films often show only an area of consolidation, cavitation with fluid level and surrounding pneumonitis is seen later. Tomography is useful to reveal the location, extent, and early evidence of cavitation. Bronchography is helpful in chronic cases to delineate local complications such as multiple abscesses and bronchiectasis.

D. Bronchoscopy: Usually indicated to rule out an obstructing bronchial lesion such as carcinoma or foreign body.

Complications.

Complications include brain abscess, massive hemoptysis, amyloidosis, and pyopneumothorax. Rupture of the abscess into the pleural space may produce tension pneumothorax. There is a sudden onset of pleural pain, dyspnea, and shock This is a grave surgical emergency requiring immediate closed chest drainage by trocar thoracotomy

Treatment

A. Medical Measures:

1. Antibiotics - In acute abscess, give high doses of penicillin and streptomycin in order to minimize lung destruction while sensitivity tests are being done Over 75% of acute lung abscesses respond favorably to intensive antibiotic therapy. The response in chronic abscess is less satisfactory, but antibiotics usually reduce the pulmonary infection to a significant degree

2. Postural drainage.

3. Bronchoscopic aspiration to promote drainage in selected cases

4. High-protein, high-caloric diet with supplementary vitamins Correct anemia by transfusions if necessary.

B. Surgical Treatment:

1. Acute abscess - Medical measures should be given a thorough trial before surgery is considered. Evidence of satisfactory progress includes decrease in cough, sputum, fever, and toxicity, and radiologic evidence of diminishing pulmonary infiltration and cavitation. If improvement has not occurred within 10-21 days, surgical drainage should be considered. This can be done under local anesthesia. A large tube is inserted into the abscess cavity through the bed of a resected rib. Adequate drainage often produces a dramatic clinical response.

2. Chronic abscess - Although simple drainage of an acute abscess usually produces good results, chronic abscess which is unresponsive to medical management requires resection of the involved segment or lobe. Preoperatively,

infection should be optimally controlled by antibiotics, postural drainage, and general supportive therapy   The bronchial tree in the region of the abscess should be carefully mapped by bronchography, and bronchoscopy should be performed.   These 2 examinations are done to explore the possibility of foreign body or neoplasm.   Neoplasms should be treated as indicated after infection has been controlled sufficiently so that surgery is feasible.

BRONCHIECTASIS

Bronchiectasis is a disorder characterized by tubular or saccular dilatation and chronic infection of the distal bronchial tree. Most clinical cases are probably secondary to focal pneumonitis occurring in childhood during an attack of pertussis, measles, scarlet fever, or other infection. Obstruction of the bronchi by foreign body is an occasional cause   Tuberculosis is frequently associated with bronchiectatic change, but since the lesion commonly is in the upper lobe the bronchial dilatation usually remains asymptomatic. Classical chronic bronchiectasis is a disease largely of the basal segments of the lower lobes, the lingula, and the right middle lobe. Congenital cystic disease of the lung must be considered when saccular dilatation is extensive or present in an unusual location.

Clinical Findings.
   A. Symptoms and Signs: There is usually a chronic cough productive of much purulent sputum, more marked on arising in the morning. Recurrent attacks of pulmonary infection, often dating back to childhood, with fever, malaise, and increased cough and sputum, are also characteristic   Sinusitis is occasionally present. Hemoptysis occurs at some time in about half of cases and may be severe.

   Significant bronchiectasis is almost always associated with coarse moist rales over the involved segments. Bronchial breathing and other chest findings are related to the extent of parenchymal involvement   Clubbing occurs in severe cases.
   B. Laboratory Findings· Bacteriologic studies of the sputum always reveal mixed infection, usually predominantly streptococci and staphylococci. Antibiotic sensitivity tests should be done. Acid-fast infection should be ruled out by smears and, in questionable cases, by culture.
   C. X-ray Findings: Plain films of the chest usually show increased bronchial markings and a variable degree of peribronchial in-
'   filtration, but changes may be minimal or absent. Bronchograms should be performed on all suspected cases. The entire bronchial tree must be mapped if surgery is planned so that all areas of involvement can be identified. Sinus films should be obtained as indicated.
   D. Bronchoscopy should be performed.

Treatment.
   A. Medical Measures: Adequate rest, good nutrition, avoidance of respiratory infections, correction of associated conditions

such as chronic sinusitis, and postural drainage are the most effective measures. A warm, dry climate is usually beneficial but not curative. Dusty, smoke-filled atmospheres should be avoided. At the onset of a respiratory infection, the patient with bronchiectasis should receive rest, symptomatic treatment, and, if an exacerbation of the bronchial infection is imminent, antibiotic therapy.

1 Antibiotics - Antibiotic therapy has no lasting effect, but suppresses cough and sputum production for a time Antibiotics are of greatest value preoperatively, during acute exacerbations in mild cases, or for patients who are not good candidates for operation. Sensitivity studies are indicated

2 Aerosol therapy - A wetting agent or bronchodilator may be administered by aerosol, preferably in conjunction with positive-pressure breathing (see p. 54).

B Surgical Treatment. Pulmonary resection is indicated in young patients in good condition who have chronic or recurring symptoms Fairly extensive areas of involvement can be removed on both sides in properly selected cases Patients up to 60 years of age with severe symptoms (especially recurrent hemorrhage) from predominantly unilateral disease are acceptable for surgery if they are good surgical risks

Preoperative preparation consists of antibiotics, aerosol therapy, and postural drainage for the purpose of minimizing pulmonary infection and decreasing sputum The daily volume of sputum should be recorded as a guide to progress It may be of interest to assess improvement by repeated tests of pulmonary function (see p 196). Operative risk and postoperative complications are materially reduced by careful preparation of patients with bronchopulmonary suppuration.

PULMONARY TUBERCULOSIS

Tuberculosis is a systemic medical disease in which surgical measures occasionally serve as useful adjuncts to other forms of treatment. It is frequently necessary to carry out diagnostic procedures for tuberculosis in surgical patients who have suspicious lesions on chest x-rays.

Establishing the Diagnosis of Pulmonary Tuberculosis.

A Symptoms and Signs· Because early tuberculosis (and even, in some cases, advanced lesions) may be essentially asymptomatic, the routine chest film is an important screening measure prior to major surgery. The characteristic symptoms of cough, weight loss, night sweats, or hemoptysis in established tuberculosis make the diagnosis much less easy to miss. When tubercle bacilli cannot be demonstrated in the sputum, it may occasionally be necessary to rely on such presumptive evidences of activity as presence of a cavity, night sweats, blood-streaked sputum, weight loss, easy fatigability, pleurisy with or without effusion, and increased erythrocyte sedimentation rate. In these cases, chest x-rays and sputum examinations should be

repeated monthly for several months and medical consultation should be obtained.

Note: Elective surgical procedures which are not directed toward the control of the tuberculous lesion should always be deferred until active tuberculosis is excluded or adequately treated.

B. Laboratory Findings
   1. Tuberculin test - Administer First Strength PPD (purified protein derivative) intradermally; if the reaction is negative, give Second Strength. A negative reaction to Second Strength PPD practically excludes a diagnosis of tuberculosis. Old tuberculin (O T.) may be used if PPD is not available
   2 Demonstration of tubercle bacilli - Recovery of tubercle bacilli is the only certain method of establishing the presence of active tuberculosis. This should be attempted by the following methods:
      a. Sputum examination - Simple smears are examined first. If these are negative, 3 or more concentrated 24 hour sputum samples should be studied by direct smear and culture or guinea pig inoculation
      b. Gastric lavage - If sputum cannot be obtained, or if further study is indicated in spite of negative sputum examinations, 2 or more fasting morning specimens of the gastric contents should be cultured or inoculated into guinea pigs.
      c. Other methods - These include the laryngeal swab or bronchial lavage technics for obtaining culture material. If bronchoscopy is done, introduce 10-15 ml of normal saline through the bronchoscope and recover by aspiration. This sample can be examined by smear and culture and is also suitable for cytologic study for tumor cells

C. X-ray Findings: Full-sized diagnostic chest films will almost always demonstrate a suspicious pulmonary lesion if active tuberculosis is present. Special radiologic studies such as lordotic views and tomograms are usually advisable for better delineation of infiltrates and cavities

Surgical Measures in Pulmonary Tuberculosis.

A. Preoperative Care. Patients with active tuberculous lesions for which surgical treatment is contemplated should have a period of rest and a course of antituberculous drug therapy preoperatively. In selected cases, especially those with recent cavities, pneumoperitoneum may be of benefit. The time devoted to these measures may vary from a few months to several years. In general, it is preferable to delay major thoracic procedures until pulmonary lesions have maximally regressed ("stabilized") as indicated by physical signs and chest films. The systemic reaction should have subsided, as indicated by disappearance of malaise, weakness, and fever, and by the return of the erythrocyte sedimentation rate to normal. Additional signs of improvement include gain in weight, marked decrease in cough and sputum, and disappearance of tubercle bacilli from the sputum.

B. Surgical Treatment: Two types of surgical procedure are widely used in tuberculosis patients who fail to respond to

medical treatment: pulmonary resection and thoracoplasty
The selection of patients, the timing of the operation, and
other details of management require the close cooperation of
internist and surgeon

1. Pulmonary resection - Patients with active pulmonary
    lesions should usually receive at least 6-8 months of chemo-
    therapy and supportive care before operation   If the sputum
    is negative, segmental and subsegmental resections are
    done to conserve lung tissue when possible. If sputum is
    positive, lobectomy is associated with fewer tuberculous
    complications than segmental resection. The role of pulmo-
    nary resection in tuberculosis may be modified as confidence
    grows in the long-range effectiveness of chemotherapy. At
    present the following indications for resection are generally
    accepted.

    a Localized disease with persistently positive sputum.
    b. Open cavity, with or without positive sputum
    c Localized nodular disease, especially when tumor is
       suspected   Opinion differs on the necessity for removing
       multiple small nodular foci   Solid lesions representing
       filled-in cavities, associated with positive sputum or
       larger than 2 cm. in size, should be resected   The
       treatment of other types may be individualized.
    d. Destroyed lung - Extensive fibrosis, bronchiectasis, and
       secondary infection are usually present, and pneumonec-
       tomy is often required
    e Thoracoplasty failure - Any of the above conditions may
       account for the failure of thoracoplasty. Laminagraphy,
       bronchography, and bronchoscopy may be necessary to
       determine the extent of residual disease.

2   Thoracoplasty - This operation and its various modifications
    are now used with decreasing frequency. Current indications
    are as follows·
    a. Chronic cavitary lesions in which resection is not feasible
        and thoracoplasty can be tolerated.
    b. Certain cases in which thoracoplasty is used to improve
        the patient's condition for a later resection.
    c   To reduce pleural "dead space" after a large resection
        and thus minimize over-distention of remaining lung tis-
        sue
    d. To close chronic empyema spaces

Postoperative Tuberculous Complications.

A Following Pulmonary Resection: •
   1. Bronchopleural fistula - This is the most frequent serious
       complication and the commonest cause of postoperative
       death. Fistula is often followed by the development of tuber-
       culous empyema. The activity of the patient's disease and
       the type of resection have an important bearing on this com-
       plication. In patients with negative sputum preoperatively,
       the incidence of fistula is about 5%. When sputum is positive,
       fistulas occur in 10-15% of cases. The highest incidence
       (15-20%) is noted in patients with positive sputum who have
       segmental resection. Bronchopleural fistula after pneumo-
       nectomy is accompanied by a mortality of about 50%.

2 Postoperative spread - This complication occurs in 5-10% of patients and can be minimized most effectively by preoperative conversion of sputum to negative   The influence of operative position on this complication is difficult to assess. Every effort should be made during and after surgery to keep the tracheobronchial tree clear of secretions by aspiration and supervised cough

B Following Thoracoplasty: The commonest complication is spread of the disease, which occurs in about 6% of cases   In general, thoracoplasty is well tolerated with relatively few complications considering the fact that most of these patients have advanced disease.

Prognosis.

Operative risk is definitely increased by a positive sputum The operative mortality for resection is about 3%, with an additional late mortality of around 2%   Relapses or persistent active disease affect a further 10%   The over-all results 5 years after resection are about 85% favorable

Thoracoplasty mortality is 1-2%, and the long-term satisfactory results are 75-80%. Prognosis is best when the operation results in sputum conversion and cavity closure

NOCARDIOSIS

Nocardiosis is caused by a variety of aerobic fungi belonging to the genus Nocardia (e g , N. asteroides, N madurae)   The infection may resemble actinomycosis or tuberculosis   The diagnosis can only be made by culture of sputum, empyema fluid, blood, or tissue   Distribution is world-wide   Treat with sulfadiazine alone or in combination with sulfamerazine (see p   618)   Continue drug therapy for 3-4 months after apparent cure

COCCIDIOIDOMYCOSIS

This disease, also known as valley fever, is caused by Coccidioides immitis, which is endemic in certain regions of the Southwest United States, Mexico, and Central and South America, with sporadic cases in Italy and Hawaii   The highly infectious organisms (arthrospores) are carried on wind-borne dust and inhaled. Even very brief exposure may produce the disease, and persons travelling through endemic areas may be infected

Clinical Findings.

A. Primary Form: The infection frequently occurs without symptoms and is detected only by the conversion of the coccidioidin skin reaction from negative to positive. Clinical manifestations in overt cases (suggestive of bronchopneumonia) include fever, cough, erythema nodosum, and pleurisy with effusion. X-ray of the lungs during the primary disease, which may last a few weeks, shows patchy soft infiltration; as this clears, residual nodules or thin-walled cavities with little surrounding infiltration may persist. Such cavities may resemble emphysematous blebs or congenital cysts. When marked coccidioidal infection of the cavity is present, the wall may be relatively thick. In the acute stage, the organisms may be found in sputum cultures. The coccidioidin skin test usually becomes positive after 10-14 days and remains positive for years. Precipitating antibodies, as determined by the precipitin test, usually appear in the course of primary infection and tend to disappear with healing. The complement fixation test often shows a low titer in primary infection with a tendency to increase in progressive disease.

B. Chronic or Granulomatous Form: 0 2% of all primary cases progress to the granulomatous stage to involve the lungs, chest wall, and other structures. Diagnosis is made by finding the organisms in infected tissues or discharges. Prognosis was poor in the past but has been improved since the advent of amphotericin B (Fungizone®) therapy.

Treatment.

Surgery is required chiefly in the treatment of the residual pulmonary nodule (coccidioidoma) or cavity left behind by primary coccidioidomycosis. Coccidioidoma usually does not require resection unless it is 2 cm. or more in diameter or unless serial films reveal that the lesion is enlarging. Radiologically, these granulomas appear as coin lesions and may be indistinguishable from carcinoma. They must be resected for diagnostic reasons unless the clinical course, previous chest films, and coccidioidin skin test establish the diagnosis of coccidioidomycosis.

Coccidioidal cavities which persist longer than 1 year should be resected to prevent complications such as secondary infection and hemorrhage. Because wedge and segmental resections may cut across infected tissues and spread the infection, lobectomy is preferred unless the process is well localized.

Encouraging results have been reported with the use of amphotericin B (Fungizone®) in the treatment of coccidioidal disease, particularly the disseminated form. This agent may also prove to be of value in the prevention of spread when infected tissue is traversed in the course of pulmonary resection. Various other drugs have mild fungicidal properties but very little clinical effect.

ECHINOCOCCUS CYST OF THE LUNG

Echinococcus (hydatid) cyst of the lung is the larval stage of the dog tapeworm, Echinococcus granulosus. Human infection occurs from ingestion of material contaminated with dog feces containing tapeworm eggs, which hatch in the intestine; the larval embryos then penetrate the bowel wall and disseminate via the blood stream. The larvas may come to rest and develop into hydatid cysts in any part of the body, but the liver (70%) and lungs (20%) are most commonly affected.

Hydatid cyst of the lung may be asymptomatic but usually causes cough, minor hemoptysis, sputum, chest pain, and fever by local pressure or by rupture with secondary infection X-ray shows a sharply defined round or oval density in the lung field. A small crescent of air is sometimes seen between the cyst and the surrounding lung and is pathognomonic. A ruptured, infected cyst has the appearance of a lung abscess. Hydatid cysts are usually solitary, but may be multiple. They usually grow slowly and occasionally reach 15 cm. or more in diameter. They are most commonly confused with neoplasm or lung abscess.

All hydatid cysts should be removed surgically if the patient's general condition permits. The mortality is negligible. The preoperative diagnosis of hydatid cyst is extremely important because rupture of an unsuspected cyst at operation may result in anaphylactic shock or in spillage of echinococcus scolices onto the pleura or wound with subsequent secondary cyst formation.

PULMONARY ARTERIOVENOUS FISTULA

Pulmonary arteriovenous fistula or aneurysm is a shunt between a pulmonary artery and vein; less frequently, the aneurysm is fed by a bronchial artery or branch from the aorta. This relatively rare anomaly may be hereditary and may be a manifestation of hereditary hemorrhagic telangiectasis (Rendu-Osler-Weber disease), which is transmitted as a simple dominant. Multiple fistulas may be present. When the shunt is sizable, unoxygenated blood bypasses the pulmonary capillaries in sufficient quantity to produce cyanosis.

Clinical Findings.
Small shunts cause no symptoms and may be discovered on a routine chest film. Fistulas may be progressive; most of them cause no symptoms until adult life. The clinical manifestations of large fistulas include dyspnea, cyanosis, clubbing, hemoptysis, and chest pain. There may be continuous or systolic murmur with or without a thrill over the aneurysm. Epistaxis and telangiectases of the skin and mucous membranes are frequently noted, and nervous aberrations may occur. Paradoxical emboli with hemiplegia and death have been reported.

When arterial oxygen saturation is below normal, polycythemia develops. Diagnosis can often be made from the appearance on x-ray of a rounded, dense shadow connected to the hilum by a broad vascular band. Most lesions occur in the lingula and the middle and lower lobes. They are multiple in 50% of cases. Angiocardiography, including study of both lung fields, is necessary for definitive diagnosis and to detect the presence of fistulas which cannot be visualized on plain films.

Treatment.

If the patient's general health is satisfactory, pulmonary arteriovenous fistulas should be excised (even though asymptomatic), since there is significant danger of progression of the lesion and of such complications as severe hemoptysis or hemothorax. Moreover, there is the threat of paradoxical cerebral emboli or brain abscess.

Surgical excision of the fistula should be accomplished with maximum preservation of pulmonary tissue and care to remove multiple lesions. Persistence or later development of cyanosis or decreased arterial saturation indicates the presence of another aneurysm.

EMPHYSEMATOUS BLEBS AND BULLAE

Air cysts in the lungs usually occur as a local exaggeration of a diffuse emphysematous process. The cause is not known, but bronchial obstruction and advanced age with loss of the elasticity of the alveolar walls may all be contributing factors. Individual alveoli are stretched and interalveolar septa are destroyed, so that the confluence of many alveoli forms cystic spaces.

Emphysematous blebs or bullae cause symptoms (1) by becoming distended with air and compressing the remainder of the lung tissue; (2) by rupturing to cause spontaneous pneumothorax; or, rarely, (3) by causing intramural hemorrhage (which on x-ray is easily mistaken for tumor). Small asymptomatic emphysematous blebs are common incidental findings, especially in the older age group. On x-ray, the bullae appear as translucent areas, usually traversed by fine linear markings. Bronchography may be necessary to delineate the bronchial patterns and determine the presence or absence of bronchiectasis.

Treatment.

Asymptomatic small blebs and bullae usually require no treatment.

A. Medical Measures: When the symptoms are mild or when diminished pulmonary reserve contraindicates surgery, medical treatment is advisable. This may include prevention and early treatment of respiratory infections, avoidance of smoking or inhalation of irritant or allergenic materials, and the use of bronchodilator drugs (see p. 54). Oxygen is administered for the relief of dyspnea or cyanosis.

B. Surgical Treatment: Large bullae causing significant symptoms or compression of lung tissue should be removed. Lesser surgical procedures than excision are generally reserved for unusual circumstances and poor-risk patients. If a large cyst

becomes acutely tense, aspiration with a needle may be life-saving. Suction drainage by a catheter placed in the cyst cavity with a trocar (Monaldi procedure) may be of value in a severely ill patient.

Prognosis.
The results of excision depend upon the condition of the remainder of the lung. When relatively large areas of normal lung exist and symptoms are due primarily to its compression by giant bullae, excision will usually restore pulmonary function to satisfactory levels.

SPONTANEOUS PNEUMOTHORAX

Spontaneous pneumothorax is usually caused by the rupture of a small emphysematous bleb (see p. 219).

Clinical Findings.
Spontaneous pneumothorax occurs most frequently in healthy young adult males. The past history is usually negative, but occasionally there is a history of recurrent pneumothorax. Onset is usually acute and characterized by severe chest pain, cough, and dyspnea. Physical signs are proportional to the extent of pneumothorax and consist of hyperresonance and diminished to absent breath sounds on the involved side. Diagnosis is confirmed by chest film.

Treatment.
A small pneumothorax (less than 25% collapse) will absorb completely within a few weeks unless the air leak in the lung continues. The patient need not be at bed rest during this period, but his activities should be limited. It may be elected to allow the air to absorb spontaneously or to obtain more rapid expansion by needle aspiration, usually through the second interspace anteriorly.

Large pneumothorax (greater than 25% collapse) should be treated by some form of suction drainage of the pleural space in order to expand the lung rapidly and thereby reduce morbidity. A small plastic tube may be introduced into the chest through a needle in the second interspace, or a catheter (e.g., No. 16 F.) may be inserted through a trocar (see p. 201). Constant suction (−25 cm. of water) is applied to the tube or catheter and immediate expansion of the lung will usually occur. Suction is continued until lung expansion is shown on x-ray and the tube or catheter is blocked by the lung. Waterseal drainage is then provided for another 24 hours; at the end of this time, if x-ray shows full expansion, the tube or catheter is removed. The entire process of expansion usually requires only 3-5 days.

Recurrent pneumothorax, either ipsilateral or contralateral, occurs in approximately 20% of cases. If 2 or more episodes have occurred on 1 side, thoracotomy should be considered in order to excise blebs and create a pleural symphysis. Thoracotomy is also occasionally necessary to control pneumothorax associated with bleeding or persistent air leak.

TENSION PNEUMOTHORAX

Tension pneumothorax occasionally develops as a result of a check-valve type of leak in the lungs. Complete collapse of the lung occurs rapidly, and the mediastinal structures are shifted to the opposite side as intrathoracic tension rises. When positive pressure rises above 15-20 cm. of water, venous return to the heart is impeded and circulatory collapse results unless the tension is relieved immediately. Physical signs include marked dyspnea, cyanosis, shift of the trachea and the apical impulse of the heart to the opposite side, and tympany to percussion and absent breath sounds on the involved side. Treatment must usually be instituted promptly without waiting for x-rays of the chest.

When tension pneumothorax occurs, a needle should be immediately inserted into the chest through the second or third interspace anteriorly. Air will rush out under pressure and relieve the tension. The needle should then be aspirated with a syringe or connected by a tubing to waterseal drainage until a catheter can be introduced into the chest as described above.

PRIMARY LUNG CARCINOMA

Bronchogenic carcinoma is the commonest primary tumor of the lung and is one of the major causes of death from cancer in men. It is 8 times more common in men than in women. Although the peak occurrence is between the ages of 50 and 70, the disease is not rare after 30. The histologic types and the relative proportion of each are as follows: epidermoid, 57%; undifferentiated, 29%; adenocarcinoma, 11%; small (oat) cell carcinoma, 2%; and bronchiolar (alveolar) cell carcinoma, 1%.

The incidence of primary lung cancer has increased markedly in recent decades. The reasons for this are not entirely clear, but inhalation of carcinogenic compounds is doubtless in part responsible. Susceptibility is greater in cigarette smokers and in people living in highly industrialized communities where the air is contaminated with smoke and fumes. A more definite etiologic relationship has long been known to exist between lung cancer and the inhalation of radioactive ore in the cobalt mines of Saxony.

Clinical Findings.

A. Symptoms and Signs: Early lung carcinoma is asymptomatic and can only be detected by x-ray. Lesions within the parenchyma of the lung may remain silent and will sometimes grow very slowly over a period of several years.

Manifestations of lung cancer are quite varied, depending upon the location and extent of the tumor. The commonest initial complaint is cough, frequently accompanied by a small amount of sputum which may be blood-streaked. Clubbing of the fingers occasionally is present in relatively early lesions.

Obstruction of a small or medium-sized bronchus is quite common, producing a localized wheeze, atelectasis, or obstructive emphysema. Physical signs over such areas may be negligible or may consist chiefly of diminished breath sounds. Acute pneumonitis frequently develops in the partially blocked

segments and not infrequently is the earliest clinical evidence of malignancy. For this reason all adults with pneumonitis should have a chest x-ray, and follow-up films should be obtained to prove complete clearing of the process.

Pneumonitis occasionally progresses to abscess formation, or a bulky tumor may break down to form a necrotic cavity. The resulting appearance may be that of a pyogenic lung abscess, and the underlying malignancy may be overlooked unless investigation is complete.

Pleural effusion is a common finding in tumors close to the visceral pleura. The effusion is frequently bloody and may contain cancer cells.

Direct extension beyond the lung may involve (1) the chest wall, causing localized or intercostal nerve pain; (2) the brachial plexus in the superior pulmonary sulcus, causing shoulder and arm pain (Pancoast's syndrome); (3) the phrenic nerve, causing paralysis of the diaphragm; or (4) the recurrent laryngeal nerve, usually at the aortic arch on the left, causing cord palsy and hoarseness.

Lymphatic spread is primarily to the hilar and mediastinal nodes, but involvement of the paratracheal and supraclavicular nodes is common. Distant, blood-borne metastases, particularly to the brain, liver, adrenals, and bones, occur frequently in advanced lesions and may occasionally be the first sign of a silent lung primary.

B. Laboratory Findings:

   1 Cytologic examination of sputum - Papanicolaou studies of sputum are positive in 50-75% of patients with lung cancer. The results of sputum examination seldom influence the decision to operate except in a select group of poor-risk patients with questionable lesions in whom a positive diagnosis justifies operation in spite of the hazard. A negative sputum examination is meaningless, and false positives occur.

   2. Scalene node biopsy - The anterior scalene and paratracheal nodes are removed for diagnostic purposes in obscure lesions and to determine operability in moderate-sized and large tumors. In left upper lobe lesions, the biopsy is performed on the left; in left lower lobe or right lung lesions, the biopsy is done on the right. In bronchogenic carcinoma without palpable nodes, the biopsy is positive in 8-10% of cases.

C. X-ray Findings:

   1. Special studies - Determination of the extent and nature of the lesion frequently requires special studies. Tomography is most often helpful. Bronchography is occasionally useful to delineate the smaller bronchi. Angiocardiography is of value in deciding the operability of medially placed tumors.

   2. Differentiation of primary from metastatic neoplasm - Primary neoplasms elsewhere in the body, especially the kidneys, thyroid, testes, and breast, occasionally produce lung metastases indistinguishable from primary lung cancer. An intravenous urogram is advisable in all operable lung tumors to rule out a primary renal tumor.

3. The solitary pulmonary nodule - Solitary pulmonary nodules are often incidental findings on chest films. These "coin" lesions are usually asymptomatic, located within the parenchyma, and associated with no distinctive physical or laboratory findings. Calcification is noted in about 50% of benign lesions and about 14% of malignancies. After complete work-up, including a review of previous films, the diagnosis is frequently unsettled. Under these circumstances thoracotomy is indicated, since about 25% of coin lesions prove to be primary lung cancer. The remainder are granulomas (42%), chiefly residuals of histoplasmosis or tuberculosis; hamartomas (8%), metastatic tumors (6%), bronchogenic cysts (4%), or 1 of a variety of rarer lesions.

D Bronchoscopy: This examination should be performed on all except discrete, peripherally located lesions. Biopsy of abnormal tissue and bronchial washings should be submitted for pathologic study. A positive diagnosis can be made by these methods in about 75% of patients. The location of the lesion in the bronchial tree often determines operability.

E Other Examinations: When pleural fluid is present it should always be examined cytologically before operation. Needle or open biopsy of the pleura under local anesthesia occasionally is diagnostic in obscure cases. Needle biopsy of the pulmonary lesion itself should only be performed to obtain a cytologic diagnosis in categorically inoperable patients.

Treatment.

Resection is the only curative treatment.

A. Indications for Thoracotomy: In the absence of evidence of inoperability (see below), all patients with proved or suspected lung cancer should be explored. Because neoplasms often remain stationary in size for many months, failure of the lesion to grow during a short period of observation does not rule out tumor.

B. Contraindications to Thoracotomy:
1. Inadequate pulmonary reserve or poor general health.
2. Evidence of distant metastases.
3. Cancer cells in pleural fluid or pleural biopsy. Bloody pleural fluid usually indicates inoperable carcinoma (or tuberculosis).
4. Positive scalene node biopsy.
5. Recurrent nerve involvement.
6. Phrenic nerve involvement usually indicates inoperability.
7. Brachial plexus involvement (Pancoast's syndrome) practically always signifies inoperability.
8. Angiocardiographic evidence of invasion or compression of the main pulmonary artery or vein at the hilus.
9. Unresectable involvement of the chest wall.
10. Bronchoscopic evidence of involvement of the proximal main stem bronchi or trachea.

C. Palliative Therapy: Roentgen therapy may be of value in the relief of pain, cough, and bronchial obstruction. However, the average survival after x-ray treatment is only about 9 months. If a patient with a small lesion refuses operation or is unsuitable for surgery, intensive supervoltage irradiation offers a faint hope of cure.

Prognosis.

Only 8-10% of all patients with carcinoma of the lung survive
5 years. However, asymptomatic patients who are operated on
solely on the basis of an abnormal x-ray have a five-year survival
rate of 40-50%. This illustrates the importance of routine chest
x-rays. The five-year survival rate for all patients undergoing
resection for carcinoma of the lung is about 25%. The prognosis is
less favorable if lymph node involvement or blood vessel invasion
has occurred. Epidermoid carcinoma has a much better prognosis
than the other cell types. Operative mortality is 5-10%.

METASTATIC NEOPLASM OF THE LUNG

Metastatic sarcoma and carcinoma of the lung are almost with-
out exception incurable. However, when a slow-growing, solitary
pulmonary metastasis appears as the only evidence of persisting
disease more than 1 year after the presumed total extirpation of
the primary lesion, consideration may be given to local excision of
the pulmonary focus. The five-year survival rate in collected se-
ries is about 12% for sarcoma and 5% for carcinoma. Many who
respond well to surgery do so because they have slow-growing
tumors, but in occasional cases a very gratifying long-term result
is obtained.

OTHER PULMONARY NEOPLASMS

Hamartomas comprise about 8% of solitary pulmonary nodules.
They are usually small, located in the periphery of the lung, and
are almost invariably benign (a few malignant hamartomas have
been reported). They are 4 times more common in men. Hamar-
tomas are embryologic remnants consisting largely of cartilage
containing variable quantities of epithelial, adipose, or muscular
tissue. Calcification may be present. These lesions must be ex-
cised for diagnostic purposes.

Rare primary pulmonary tumors, including leiomyoma, leio-
myosarcoma, and lymphoma, are generally indistinguishable from
bronchogenic carcinoma. The diagnosis is usually made only by
histologic examination.

BRONCHIAL ADENOMA

Bronchial adenoma occasionally invades locally or metasta-
sizes, but its behavior is different from that of bronchogenic car-
cinoma. Histologically there are 2 characteristic patterns: the
carcinoid and cylindroid forms, with transitional and variant types
The carcinoid form resembles carcinoid tumors of the gastrointes-
tinal tract and usually involves one of the major bronchi. Cylin-
droid adenoma tends to occur near the carina or in the trachea,
and typically shows relentless local invasion. Both types of ade-
noma grow slowly, are more common in women, and tend to occur
earlier than bronchogenic carcinoma (mean age 40). Metastases
occur in about 15% of cases.

Dry cough, unilateral wheeze, and recurrent hemoptysis are characteristic in the early stage. The average duration of symptoms is over 3 years, and the late changes are due chiefly to bronchial obstruction and pulmonary infection. Findings are often distinguishable from bronchogenic carcinoma only by their chronicity. Most patients show x-ray changes suggesting atelectasis or inflammatory infiltration. Bronchoscopic examination gives the correct diagnosis in less than half of patients.

Bronchoscopic resection is not a reliable method of treatment unless the circumstances are exceptionally favorable. Cure usually requires resection of the involved bronchus, adjacent nodes, and damaged lung tissue. Removal of a small adenoma by bronchotomy may occasionally be feasible

The five-year survival rate after resection is about 80%.

DISEASES OF THE MEDIASTINUM

TUMORS OF THE MEDIASTINUM

Mediastinal masses consist chiefly of a variety of neoplasms and cysts which tend to occur in characteristic sites (see p. 226). The most frequently encountered tumors are thymomas and teratomas in the anterior mediastinum; lymphomas in the anterior and middle mediastinum; and tumors of the intercostal nerves, sympathetic nerve trunks, and ganglia in the posterior mediastinum Other conditions which must occasionally be considered in the differential diagnosis of mediastinal masses include cardiovascular abnormalities (especially aneurysm), diaphragmatic hernias, esophageal and pulmonary lesions, granulomas (histoplasmoma, tuberculoma, and sarcoid), hydatid cyst, and metastatic malignancy.

Clinical Findings.

About half of mediastinal tumors are discovered on mass survey or routine chest x-ray, and the majority of these are asymptomatic. Specific symptoms and signs depend upon the size, location, and nature of the tumor. Cough, chest pain, and dyspnea are among the commonest complaints. Physical signs are frequently absent or minimal. The following are of particular interest when present: cervical or generalized lymphadenopathy; venous congestion in the head, neck, and upper extremities (superior vena caval obstruction); hoarseness (recurrent nerve palsy); Horner's syndrome (involvement of the cervical sympathetic nerves); and elevation of the diaphragm (phrenic nerve paralysis). Nerve damage is usually caused by inoperable malignant invasion. When a malignant mediastinal tumor is suspected, a careful search should be made for evidence of primary or metastatic neoplasm outside the mediastinum.

Laboratory examinations include routine chest films, tomography, and, if a vascular lesion is suspected, angiocardiography. Blood and bone marrow studies for blood dyscrasias are indicated if a lymphoma is suspected. Sputum smears, cultures, and cytologic studies may be indicated. Determination by the scanning technic of radioactive iodine uptake by the mediastinal mass is

Location of Tumors and Cysts in the Mediastinum

Anterior Mediastinum	Superior Mediastinum	Middle Mediastinum	Posterior Mediastinum
Thymoma	Goiter	Bronchogenic cyst	Neurilemmoma
Thymic cyst	Bronchogenic cyst	Lymphoma	Neurofibroma
Teratoma	Parathyroid adenoma	Pericardial cyst	Ganglioneuroma
Goiter	Myxoma	Plasma cell myeloma	Sympathico-blastoma
Parathyroid adenoma	Lymphoma		Fibrosarcoma
Lymphoma			Lymphoma
Lipoma			Goiter
Fibroma			Xanthofibroma
Lymphangioma			Gastroenteric cyst
Hemangioma			Chondroma
Chondroma			Myxoma
Rhabdomyo-sarcoma			Meningocele
			Paraganglioma
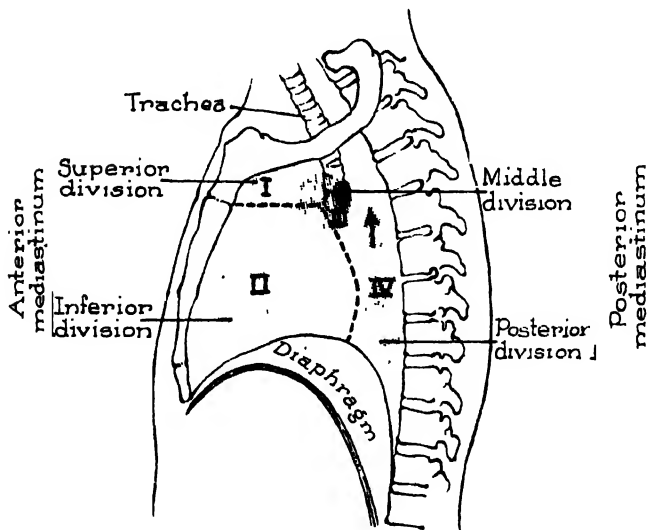
Divisions of the Mediastinum

occasionally diagnostic in substernal goiter Scalene and para-
tracheal node biopsy is valuable, especially if lymphoma or other
malignancy is being considered. Direct needle biopsy of the medi-
astinal mass may occasionally be feasible in order to establish the
diagnosis in an accessible but obviously inoperable tumor. The
response of the mediastinal mass to a course of x-ray therapy has
proved an unreliable diagnostic criterion.

Treatment.

The majority of mediastinal enlargements require thoracotomy
for positive diagnosis and definitive treatment. In most cases noth-
ing is to be gained by delay of exploration when an operable lesion
cannot be ruled out. Even benign, asymptomatic lesions should
rarely be treated expectantly. Sooner or later, because of their
critical location, they usually cause serious difficulty as a result
of pressure, infection, or rupture

Characteristic Features of the Commonest Mediastinal Tumors.

A. Mediastinal Goiter: This lesion is usually a direct extension
   of a nontoxic adenomatous goiter in the neck. The usual loca-
   tion of the goiter is the anterior superior mediastinum, but
   descent into the upper posterior mediastinum does occur.

   In 50% of cases the disease is asymptomatic and is detected
   by routine chest films; in the remainder, symptoms include
   dyspnea, cough, pain, and dysphagia. The cervical portion of
   the thyroid is usually, but not always, enlarged and palpable
   The substernal thyroid is readily demonstrated by x-ray.
   Some degree of tracheal (and possibly esophageal) deviation
   and compression is typically present. The rounded, homogen-
   eous density in the mediastinum is continuous above with the
   cervical shadow, and on fluoroscopy the mass is usually seen
   to move upward with swallowing. If functioning thyroid tissue
   is present in a mediastinal goiter, the diagnosis can be con-
   firmed by administering radioiodine and taking a scanogram of
   the area Superior mediastinal enlargements should be exam-
   ined with scanography unless thyroid origin can be ruled out.

   Accurate preoperative diagnosis is especially desirable in
   superior mediastinal growths since this determines the opera-
   tive approach. Mediastinal goiters should be approached
   through the usual thyroidectomy incision except in the rare
   instance (e.g., posterior mediastinal goiter) where a combined
   thoracic and cervical approach may be required.
B. Bronchogenic and Gastroenteric Cysts: These cysts represent
   embryologic remnants or anomalies of the respiratory and ali-
   mentary tracts, respectively Bronchogenic cysts are lined
   with respiratory epithelium, and may contain smooth muscle
   and cartilage in the wall. They are most often found in the
   parahilar or right paratracheal regions. The gastroenteric
   cysts are usually closely associated with the esophagus or ac-
   tually intramural, and are lined by squamous, gastric, or in-
   testinal epithelium. Those containing gastric epithelium may
   have acid-secreting cells, and peptic ulceration of the lining
   sometimes occurs.

   Bronchogenic cysts usually become symptomatic only in
   adult life, if at all, whereas 75% of gastroenteric cysts are

diagnosed in the first year of life because of such serious complications as peptic ulceration, perforation into a bronchus or pleural cavity, or hemorrhage. The symptoms of both bronchogenic and gastroenteric cysts depend primarily on their size and location and on the presence or absence of infection. Chest pain, cough, wheezing, and slight dysphagia are the commonest complaints. When the cyst is infected the clinical appearance may resemble lung abscess or empyema. By x-ray, bronchogenic cysts are ovoid, smooth in outline, and homogeneous in density except when a bronchial fistula exists, in which case an air-fluid level can be seen. They are usually located near the midline and, because of their close relation to the trachea, bronchi, and esophagus, may be seen on fluoroscopy to move up and down with respiration or swallowing

The treatment of bronchogenic and gastroenteric cysts - even if asymptomatic - is surgical removal, provided there is no contraindication to operation.

C. Thymoma: Thymomas are usually composed of lymphocytes and epithelial cells. Since the proportion of these 2 cell types varies in different tumors, the microscopic picture is sometimes difficult to interpret; before diagnosing a tumor as primary in the thymus, one must carefully rule out teratoma, lymphoma, and metastatic tumor. Necrosis, hemorrhage, and cyst formation within thymomas are not uncommon. Cystic change is occasionally so extensive that only a fibrous capsule containing a few remnants of thymic tissue remains (thymic cysts). The histologic differentiation of benign from locally malignant variants of thymoma is difficult or impossible. About one-fourth of these lesions break through their capsule and invade locally or implant on neighboring surfaces, and in this sense they are "malignant"; however, lymphogenous or hematogenous metastases are not known to occur.

Thymoma occurs in about 15% of patients with myasthenia gravis, and about 75% of patients with thymoma have myasthenia gravis; but removal of the tumor has little or no effect on the myasthenia. Myasthenic symptoms may develop years after a thymoma has been known to be present. In females, especially those under 30 years of age, the removal of a non-neoplastic thymus gland is said to be associated with an increased remission rate in myasthenia gravis.

The average age of patients with thymoma is about 45, and there is no significant difference in sex incidence. The commonest symptoms of thymoma are those of myasthenia gravis. Chest pain occasionally occurs and may indicate invasion of the pleura. Tracheobronchial or superior vena caval obstruction sometimes develops, and hemorrhage into the tumor may produce a rapid onset of pressure symptoms. Roentgenologically, the tumor is usually round or oval and sharply delineated from the surrounding tissues. Its typical location is in the anterior mediastinum, usually anterior to the aortic arch and the base of the heart, but thymomas may be found at lower levels anteriorly or, rarely, posterior to the superior vena cava. Calcification is seen on x-ray in about 25% of tumors and does

not rule out local invasion.

Thymomas should be removed since one-fourth of these tumors are locally invasive and it is usually impossible to distinguish these from the noninvasive majority. Myasthenia gravis is the chief cause of operative mortality, and severe grades of this disease may contraindicate surgery. When the tumor is removed completely, recurrence is not likely. Irradiation of thymomas in patients with or without myasthenia gravis only occasionally results in reduction in size of the tumor.

. Teratoma: These tumors arise within the anlage of the thymus and consist of tissues derived from 2 or all 3 of the primitive germ layers. About one-third are malignant. Dermoid cyst is a type of benign, unilocular teratoma containing sebaceous material and hair and lined by stratified squamous epithelium and dermal appendages.

Teratomas may occur at any age but are most frequently seen in persons between 20 and 40. About one-third are asymptomatic; the remainder cause cough, chest pain, dyspnea, or other pressure symptoms. In rare cases the tumor ruptures into the esophagus or trachea and the patient vomits or spits up hair. Malignant lesions infiltrate locally, increase rapidly in size, and may produce superior vena caval obstruction, pleural implantation, and distant metastases.

Teratomas are characteristically precordial in location on x-ray, although they may occasionally be found in the posterior mediastinum. They are of homogeneous density, and discrete, round, and smooth in outline unless local invasion has occurred. If the mass is large it may project into either pleural cavity, causing atelectasis, and may even appear to occupy an entire hemithorax. Pleural effusion may be present, especially in malignant teratoma. When the tumor contains bone or teeth, a specific diagnosis can be made. In most cases diagnosis can be established only by exploration.

Treatment is by excision, which is curative in benign lesions. The prognosis of malignant teratoma is very poor; most patients die within 1 year after diagnosis.

. Lymphoma: This is the most frequent cause of a mediastinal mass. The most common symptoms are cough, pain, dyspnea, wheezing, hoarseness, weight loss, fatigue, and fever. Peripheral lymphadenopathy or splenomegaly may be present. The mediastinal tumor consists of involved lymph nodes, usually in the anterior or middle mediastinum. Bilateral enlargement of the mediastinum is suggestive of lymphoma, but unilateral mass does not preclude the diagnosis. The outlines of the mass on x-ray may appear rounded or lobulated and may be either sharp and clear-cut or indistinct.

Palpable peripheral nodes should be biopsied. If this is not diagnostic (in suspicious cases), the scalene nodes should be removed on 1 or both sides for histologic examination. By means of these procedures (as well as blood and bone marrow studies) a positive diagnosis can be made in over half of cases. Exploratory thoracotomy is contraindicated when the diagnosis can be established by other methods since lymphoma is invariably a disseminated disease and local manifestations are usually best managed by x-ray or chemotherapy.

F. Neurogenic Tumors: These are the most frequent primary mediastinal neoplasms. Nearly all neurogenic tumors of the mediastinum arise from the intercostal nerves or the paravertebral sympathetic trunks and are therefore usually found in the posterior mediastinum. Rarely the tumor originates in the neck and extends into the superior mediastinum. Neurilemmoma (schwannoma), a benign neoplasm of the nerve sheath of Schwann, is the most common neurogenic tumor. Many of these neoplasms have been wrongly classified as neurofibromas, which are quite uncommon in the mediastinum. Ganglioneuroma is a rare benign neoplasm of the sympathetic ganglia which may be distinguished by its tendency to reach a very large size and to grow into or out of an intervertebral foramen, producing an "hourglass" tumor which may exert pressure on the spinal cord. The benign neurogenic tumors have malignant counterparts which are fortunately quite rare (malignant schwannoma, malignant ganglioneuroma, sympathicoblastoma).

Neurogenic tumors are usually benign and asymptomatic. The usual complaint is pain referred to the chest, neck, or arm. The diagnosis is usually made on routine x-rays, which characteristically show a round, homogeneous mass with distinct borders in the posterior mediastinum. Occasionally the growth lies behind the heart shadow and is partially obscured on routine exposures. Rib or vertebral erosion may occur, and widening of the intervertebral foramen may be present in the "hourglass" lesions. Calcium can sometimes be visualised. Treatment is by excision.

G. Pericardial Cyst: These cysts probably result from a failure of fusion of 1 or more of the lacunae which merge to form the pericardial sac, but they may represent lymphatic cysts. They are benign, unilocular, and thin-walled; attached to the pericardium, and filled with clear serous fluid. They are usually asymptomatic. Most are discovered on routine chest films, which typically show a smoothly rounded, sharply demarcated tumor of uniform density lying against the pericardium, diaphragm, and chest wall in the right or left anterior cardiophrenic angle. Because of their position they may be confused with diaphragmatic hernia, dermoid cyst, or a pericardial fat pad. Surgical removal of pericardial cysts is indicated because it is not possible to distinguish them from more serious lesions.

H. Granuloma: Granulomatous inflammation of the mediastinal lymph nodes may be caused by histoplasmosis, tuberculosis, coccidioidomycosis, and sarcoidosis. The majority of patients have no symptoms directly related to the enlarged nodes, which are usually located alongside the trachea or hilum and protrude into the thorax as smoothly oval, rounded, or lobulated masses. Calcification may be present. The radiologic appearance is often typical of sarcoidosis, with pulmonary parenchymal involvement and bilateral hilar enlargement. When only unilateral hilar lymphadenopathy is present in sarcoidosis, the diagnosis is difficult. Scalene node biopsy and skin tests for tuberculosis, histoplasmosis, and perhaps coccidioidomycosis should be done. Surgical removal of granulomatous lesions is usually not necessary except for diagnostic purposes when other

measures fail.   The dense inflammatory reaction around the nodes may make excision difficult.

ACUTE MEDIASTINITIS

Mediastinal infections are chiefly due to perforation of the esophagus by instrumentation or foreign body or to leakage of esophageal suture lines after surgery.   Clinical findings usually consist of fever, tachycardia, and prostration.   Deep-seated substernal, epigastric, or neck pain may be present.   The superior mediastinum is most frequently involved.   Chest films show widening of the mediastinum and blurring of the aortic prominence, and pulmonary infiltration and bilateral pleural effusion may occur.   A swallow of a soluble contrast medium, e.g., Methylglucamine Diatrizoate, N.N.D. (Gastrografin®)(never barium), may show the site of perforation.

Minimal leakage from the esophagus after endoscopy usually responds satisfactorily to intensive antibiotic therapy and restriction of oral intake   Large tears and leaks should be repaired immediately if discovered early; otherwise, drainage by mediastinotomy is required for abscesses or spreading infection.

10 . . .

Surgery of the
Heart and Great Vessels

Both congenital and acquired cardiac lesions may, in selected cases, be amenable to partial or complete surgical correction. An accurate anatomic and functional diagnosis is essential preoperatively. In addition to routine studies, including conventional x-ray and Ecg. studies, it is frequently of value to perform cardiac catheterization or angiocardiography, particularly in complex congenital anomalies. Characteristic data from right heart catheterization of some common, surgically treatable cardiac lesions are summarized in the table on p. 239.

CARDIAC TAMPONADE DUE TO TRAUMA

Patients with large cardiac wounds die promptly. Those who reach the surgeon have small wounds, and death is usually caused by cardiac tamponade rather than exsanguinating hemorrhage. The accumulation of 150-200 ml. of blood in the pericardial sac is sufficient to prevent filling of the heart during diastole.

Clinical Findings.
A. Symptoms and Signs: If intrapericardial bleeding is slow, there may be a latent period varying from minutes to hours after a penetrating chest wound until the patient collapses. Classical features of fully-developed, acute tamponade include pallor, sweating, cyanosis, dyspnea, hypotension, distant or inaudible heart sounds, and a weak pulse which may be irregular or paradoxical (i.e., becomes weaker or disappears on inspiration). The superficial veins, especially those of the neck, may be distended. The patient is sometimes unconscious or disoriented.
B. Laboratory Findings: Venous pressure is almost invariably elevated.
C. X-ray Findings: X-ray of the chest often shows a heart shadow of normal size. Fluoroscopy usually reveals diminution or absence of cardiac pulsations.
D. Ecg. Findings: Ecg. may be normal, may demonstrate arrhythmias or a pattern consistent with pericarditis, or may show various nonspecific changes.

Treatment.
When a presumptive diagnosis of a heart wound is made, an infusion of 5% glucose in water or saline should be started immediately through a large needle or venous cutdown. Blood is prepared at once and plasma or a plasma expander is used in the meantime to control shock, if present. The semi-sitting position is often best tolerated. Morphine and oxygen are administered as indicated and the operating theater alerted while diagnostic examinations are being completed. In all cases of tamponade, aspiration of the peri-

cardium is advisable while the operating room is being prepared. If the response to pericardiocentesis is good, this procedure can be recommended as definitive treatment in selected cases.

A. Pericardiocentesis:
1. Sites of puncture - Avoid puncture of the ventricular muscle.
   a. Epigastric area (preferred) between the xiphoid and the left costal margin. Insert the needle upward at an angle of 45° and pointed toward the midline. Advance the needle slowly until blood or fluid is obtained or until a scraping sensation or movement of the needle indicates contact with the ventricular wall. The pericardium is reached at about 3-4 cm.
   b. Precordium (alternate) at the left fourth or fifth interspace 1-2 cm. within the border of cardiac fullness or x-ray silhouette. If hemopericardium is present, it should be encountered at about 3-5 cm.
2. Equipment -
   a. A No. 18 or 17 gauge needle with a short bevel, 6-8 cm. long.
   b. A No. 26 or 27 gauge (hypodermic) needle and a No. 22 gauge (6 cm.) needle to infiltrate the skin and deeper structures with procaine.
   c. A 20-30 ml. syringe. A 3-way stopcock or a 4-inch piece of rubber tubing may be used to connect the syringe to the needle.
   d. Procaine hydrochloride, 1 or 2%.
3. Technic - Apply antiseptic and drape with towels. Infiltrate the puncture site with procaine. Insert the needle slowly with the syringe attached, aspirating gently at various levels. Inadvertent puncture of the ventricle is usually harmless, particularly when the epigastric approach is used. Remove all blood or fluid from the pericardium and withdraw the needle.

B. Thoracotomy: Immediate thoracotomy is indicated (1) if the heart wound is obviously severe, as shown by rapid blood loss, irreversible collapse, or uncontrollable tamponade; or (2) if tamponade recurs soon after pericardial tap. Suture of a heart wound may be accomplished with relative ease by trained personnel when proper facilities are available; otherwise one must rely on repeated pericardiocenteses and blood replacement.

Prognosis.

The mortality in patients suitable for conservative treatment by pericardial aspiration is as low as 6% in some series (Elkin). Patients who survive heart wounds usually have no subsequent disability. Rarely, a constrictive pericarditis develops as a result of hemopericardium.

ACUTE PERICARDITIS

Acute pericarditis is of surgical significance chiefly (1) when a pyogenic infection requires open drainage and (2) when pericardial biopsy is needed to establish the diagnosis.

CLASSIFICATION, DIAGNOSIS, AND TREATMENT OF CONGENITAL HEART DISEASE*

Lesion and Hemodynamics	Diagnosis: Clinical	Diagnosis: Routine X-ray and Ecg.	Treatment and Prognosis
		ACYANOTIC TYPES	
POSITIONAL DEFECTS			
Dextrocardia Right-sided heart, with or without reversal of position of other organs. Heart may be normal or have severe defects.	Apical pulse and sounds on right side of chest. Reversal of position of other organs, i.e., situs inversus may be present.	Cardiac silhouette on right side. P and T waves inverted. QRS predominantly down in Lead I. Lead II resembles normal Lead III and vice versa.	No treatment necessary. In absence of other cardiac anomalies, prognosis is excellent.
Coronary artery anomalies One or both coronaries may arise from pulmonary artery. Single coronary may arise from aorta.	Onset of symptoms within a few days or weeks of birth. Recurrent dyspnea, pallor, sweating, vomiting, coughing, tachycardia, hepatomegaly.	X-ray shows progressive enlargement of heart. Ecg.: Inversion of T waves over left precordium, especially just after feeding.	No treatment is available. Death occurs before one year of age.
AORTIC ARCH ANOMALIES			
Coarctation of aorta "Infantile" type: Constriction proximal to ductus (incompatible with life beyond infancy). "Adult" type: Constriction at or distal to ductus arteriosus. Blood pressure high in head and upper extremities, low in lower part of body and legs.	May have minimal findings in early childhood, heart failure in infancy. Older child may have numbness of legs or signs of heart failure. Hypertension may cause headaches, epistaxis, dizziness. Absent femoral or dorsalis pedis pulsation, may have systolic murmur over sternum and left back.	Cardiac enlargement may or may not be present on routine exam. Site of coarctation and dilatation may be seen on PA view. In older child, ribs may show notching, from collateral vessels. Ecg. usually shows left ventricular hypertrophy.	Surgical excision of constricted portion of vessel and anastomosis. Unless clinical condition indicates increasing cardiac failure, operation is best carried out between the ages of 6 and 12. Prognosis is excellent.
VASCULAR RINGS Double aortic arch; right aortic arch with right brachial arch; anomalous right subclavian artery; anomalous innominate artery; anomalous left carotid artery.	Usually asymptomatic. Constriction of esophagus and trachea may cause vomiting, dysphagia, stridor, recurrent respiratory infections. Child may hold head in hyperextension.	Ecg. is nonspecific. X-ray of heart with barium in esophagus will establish diagnosis. Outlined trachea with contrast material will add to diagnostic detail.	Surgical correction if trachea or esophagus is constricted.

*Modified and reproduced, with permission, from Silver, Kempe, and Bruyn: Handbook of Pediatrics, Third Edition. Lange, 1959.

Persistent patent ductus arteriosus High aortic pressure shunts blood through ductus into pulmonary artery in systole and diastole. Inc. left vent. output, blood volume, and pulse pressure.	Murmur in 2nd or 3rd interspace to left of sternum, appearing in infancy. May be systolic alone early but by 6 years of age most patients will show diastolic component with "machinery" sound and thrill. Wide pulse pressure.	Enlargement of left atrium and ventricle, increased pulmonary vascular markings, and expansile pulsation of pulmonary arteries. Ecg. may be normal or show left ventricular or combined hypertrophy.	Surgical division or ligation of the ductus. Operative mortality is so low that procedure should be undertaken as soon as possible after diagnosis. With surgical repair, prognosis is excellent. Without repair, SBE or cardiac failure may develop.
SEPTAL DEFECTS Interatrial septal defect Septum secundum and foramen ovale defects, high in atrial septum. Septum primum defect, low in atrial septum, usually large, often associated with defect of mitral or tricuspid valve. Anomalous pulmonary venous return frequently present. Increased blood shunts from left to right atrium. Increased pulmonary vascular resistance may develop in time, particularly with primum defect. Lutembacher's syndrome - atrial septal defect with congenital or acquired mitral stenosis.	Very common congenital defect. More common in girls. Growth may be retarded and chest may bulge in time from enlarged right ventricle. Murmur is systolic. Secundum defect: Grade III blowing. Primum defect: Grade IV harsh, loudest near 2nd left interspace in secundum and transmitted to apex or back in primum defect. May have signs of arachnodactyly.	X-ray: Globular enlargement of heart, with marked enlargement of right heart. Prominent enlarged pulmonary vessels, which may show pulsation on fluoroscopy. Small aortic shadow. Ecg.: Right incomplete or complete bundle branch block is common.	Surgical closure is recommended in all cases except those with marked pulmonary vascular resistance. Secundum defects can usually be repaired under hypothermia, but open heart approach to all lesions is preferable. Without repair, life expectancy is 37 years. With early repair of secundum defects, life expectancy is probably normal.
Interventricular septal defect Usually involves membranous septum. Oxygenated blood passes from left ventricle to right. Symptoms and signs related to size of defect. With small defect, symptoms are mild or absent ("maladie de Roger"). Large defects produce marked disturbance (Eisenmenger complex, see below).	Common congenital defect often discovered on "routine" exam. Murmur: 3rd left interspace, systolic, loud, harsh, often with thrill.	X-ray: Heart size is normal in small defects. In large shunts, both ventricles and the pulmonary artery are enlarged, with pulmonary engorgement and hilar dance. Ecg.: Normal in small defects; may show either right or left ventricular hypertrophy in large defects.	Surgical closure by open heart method is advisable except in presence of marked pulmonary vascular resistance. Operation may be temporarily deferred in small, asymptomatic defects awaiting further improvement in operative technics. Optimum age for surgery is between 2 and 15 years.

CLASSIFICATION, DIAGNOSIS, AND TREATMENT OF CONGENITAL HEART DISEASE (Cont'd.)

Lesion and Hemodynamics	Diagnosis		Treatment and Prognosis
	Clinical	Routine X-ray and Ecg.	

ACYANOTIC TYPES (Cont'd.)

OUTFLOW TRACT LESIONS OF LEFT HEART

Lesion and Hemodynamics	Clinical	Routine X-ray and Ecg.	Treatment and Prognosis
Aortic stenosis Constriction of aortic valve causes left ventricle to enlarge. Systolic BP is lower than normal, and pulse pressure is low unless there is associated aortic insufficiency.	Rare lesion. Murmur is loud and harsh in 2nd or 3rd right interspace, with transmission to neck and prominent thrill. 2nd heart sound is weak. Narrow pulse pressure. Rare diastolic murmur.	X-ray may show slight left ventricular enlargement. Ecg. shows left ventricular preponderance.	Surgical valvulotomy is indicated in cases with high-pressure gradient across valve, as determined by catheterization of left ventricle. Operative mortality less than 10%. Restriction of activity will postpone failure. Prognosis guarded without surgery. Sudden death may occur. Life expectancy to 30 years.

OUTFLOW TRACT LESIONS OF RIGHT HEART

Lesion and Hemodynamics	Clinical	Routine X-ray and Ecg.	Treatment and Prognosis
Valvular pulmonic stenosis Usually occurs with other anomalies; if the sole lesion, right ventricular pressure is increased. Pulmonary blood flow is normal. With failure, cyanosis may result from diminished blood flow.	Symptoms rare in infancy. Many children asymptomatic. Dyspnea, fatigue, and, rarely, cyanosis develop after age 2. Sudden failure may occur as first sign in late childhood. Systolic murmur is harsh, loud, often with thrill in 2nd to 4th left interspaces.	Right ventricular enlargement with dilated main pulmonary artery. Pulmonary vascular markings may be diminished peripherally. Ecg. shows right ventricular hypertrophy.	Pulmonary valvulotomy. Prognosis is poor without surgical repair (death within 2-4 years after first attack of failure). Results of operation, particularly by open methods, are excellent.

CYANOTIC TYPES

INTERTRACT LESIONS

Lesion and Hemodynamics	Clinical	Routine X-ray and Ecg.	Treatment and Prognosis
Valvular pulmonic stenosis with atrial septal defect Venous blood from right atrium shunted into left atrium, thus cyanosis before oxygenation, thus cyanosis. Right atrium and ventricle enlarged.	Clinical picture may simulate tetralogy of Fallot (see below), although cyanosis is seldom as marked and may be absent at rest. Differentiation only by cardiac catheterization (see table on p. 239) or angiography.	X-ray findings similar to tetralogy of Fallot (see below), but the pulmonary vessels are more prominent. Ecg. shows right ventricular hypertrophy.	Valvulotomy of stenotic pulmonary valve and closure of septal defect by open heart surgery. Results are generally good with low mortality.

Cor triloculare Failure of development of the right ventricle, which may persist in rudimentary form.	Cyanosis at birth. Murmur systolic, over precordial area, soft, blowing. Dyspnea may be present.	Large left ventricle, extending posteriorly. Ecg.: Left ventricular hypertrophy and conduction defects.	Treat as cardiac failure. Life expectancy limited; may be slightly longer if no other cardiac anomalies.
Cor biloculare Interatrial septal defect and small right ventricle or large interventricular septal defect.	Rare lesion. Marked cyanosis, dyspnea, and cardiac failure early in life.	Similar to those of cor triloculare (above).	No treatment available. Treat as cardiac failure. Death usually within a few months.

OUTFLOW TRACT LESIONS OF RIGHT HEART (USUALLY COMBINED LESIONS)

Pulmonary stenosis with interventricular septal defect (Tetralogy of Fallot) May include overriding dextroposition of aorta. Right to left shunt is fundamental to lesion. Small blood flow through pulmonary artery. Aortic blood poorly oxygenated.	Most common cyanotic type. Cyanosis early as ductus closes ("blue baby"). Lips, nail beds may be blue. Dyspnea apparent early. Older child may squat or lie down during play. "Spells" may occur due to cerebral anoxemia. Clubbing of digits, usually after 2 years. Loud, harsh systolic murmur at left base, thrill frequent. Polycythemia, increased Hct. and RBC. Short circulation time due to overriding aorta.	X-ray usually shows typical "wooden shoe" contour of heart: heart is not enlarged, apex is blunt, elevated above diaphragm; pulmonary artery segment is concave. Right ventricle enlarged. Pulmonary vessels appear scarce, and lung fields unusually clear. Ecg. shows marked predominance of right ventricle. Peaked P waves.	Open heart repair of I-V septal defect and relief of pulmonary stenosis. Severe symptoms in an infant with a poorly developed vascular bed best palliated by a Blalock procedure (anastomosis of subclavian artery to pulmonary artery) or Potts operation (side-to-side anastomosis of aorta to pulmonary artery), a corrective operation can be attempted later. Optimum age for definitive surgery is 6-8 years if delay is feasible. Without surgery, death by age 20 is usual. Prognosis is probably markedly improved by surgery, but long-range results are not known.
Large ventricular septal defect (Eisenmenger complex) usually includes dextroposition of aorta and pulmonary hypertension resulting from these defects. Eventually produces right to left shunt and cyanosis.	Severe symptoms develop early with congestive failure, sometimes first in infancy. Dyspnea, repeated infection, and cyanosis are all common. Hemoptysis may occur in older children. Systolic murmur is not as loud as with smaller ventricular septal defects. Usually rumbling character. No thrill. Accentuated 2nd pulmonic sound.	Cardiac enlargement, apparently involving both ventricles and left atrium. Pulmonary vessels engorged, with pulsation on fluoroscopy. Ecg. shows right ventricular hypertrophy, occasional right bundle branch block.	Closure of the defect by open heart surgery may be feasible if pulmonary pressure is not too high. Inoperable cases are treated by restriction of activity and usual measures for cardiac failure as necessary.

CLASSIFICATION, DIAGNOSIS, AND TREATMENT OF CONGENITAL HEART DISEASE (Cont'd.)

Lesion and Hemodynamics	Diagnosis		Treatment and Prognosis
	Clinical	Routine X-ray and Ecg.	
		CYANOTIC TYPES (Cont'd.)	
Transposition of great vessels Aorta arises from right ventricle; pulmonary artery from left ventricle. Blood from right heart goes through systemic vessels and returns to right side. Blood from left heart goes through lungs to return to left side. Incompatible with life unless patent foramen ovale, ductus arteriosus, or interventricular septal defect also present. Coronary arteries arise from aorta, carry anoxic blood.	Marked cyanosis apparent at birth. Dyspnea, engorged neck vessels, and enlarged liver apparent in neonatal period. Murmurs not typical or always present. Heart enlarges rapidly in first few weeks of life.	Enlargement of ventricles with narrow base on PA view; base wide on lateral or oblique views. Pulmonary vascular markings increased. Ecg. may show myocardial damage due to ischemia and right ventricular hypertrophy. Angiocardiography shows the aorta emerging from the right ventricle.	Surgical treatment is still experimental but is developing rapidly. In infants the most satisfactory results have been obtained from creation of an interatrial septal defect. Attempts at shifting the veins to the proper atria have been partially successful. Anastomosis between the superior vena cava and the right pulmonary artery is a promising procedure, and the early result in a patient so treated is excellent.
Tricuspid atresia Marked stenosis of tricuspid valve, small right ventricle with or without pulmonary stenosis. Blood from right atrium goes through septal defect to left atrium and into systemic arteries. Pulmonary circulation through patent ductus arteriosus or interventricular septal defect. Collateral circulation through bronchial arteries must develop to sustain life.	Cyanosis apparent shortly after birth. Neck vessels and liver engorged and pulsating. Murmurs not typical or always present. If blood supply to lungs is by way of an open ductus, a continuous murmur is present.	Enlargement of left ventricle. Concave left border. Pulmonary vessels diminished in size. Aorta continuous with cardiac shadow in left anterior oblique views. Ecg. shows left ventricular hypertrophy. Only cyanotic type with this finding.	Surgery has been attempted in some cases, with pulmonary anastomosis as in tetralogy. Slight improvement noted. Prognosis is poor; these patients rarely survive 1-3 months.

RELATIVE PRESSURES AND OXYGEN CONTENT AS OBTAINED DURING CARDIAC CATHETERIZATION
(Modified after Marple)*

	PRESSURE			OXYGEN CONTENT			
	Right Atrium	Right Ventricle	Pulmonary Artery	Right Atrium	Right Ventricle	Pulmonary Artery	Systemic Artery
Normal heart	5/0	25/2	25/8	Equals vena cava	Equals right atrium	Equals right ventricle	95-100% saturated
Atrial septal defect	Normal or slightly increased	Normal or increased	Normal or increased	Greater than vena cava	Equals right atrium	Equals right ventricle	Normal†
Ventricular septal defect	Normal	Normal or increased	Normal or increased	Equals vena cava	Greater than right atrium	Equals right ventricle	Normal†
Patent ductus arteriosus	Normal	Normal or increased	Normal or increased	Equals vena cava	Equals right atrium	Greater than right ventricle	Normal†
Tetralogy of Fallot	Normal	Increased	Decreased; lower than right ventricle	Equals vena cava	Equals or is greater than right atrium	Equals right ventricle	Decreased
Eisenmenger complex	Normal	Increased	Increased	Equals vena cava	Greater than right atrium	Equals right ventricle	Decreased
Valvular pulmonary stenosis	Normal	Increased	Decreased	Equals vena cava	Equals right atrium	Equals right ventricle	Normal
Valvular pulmonary stenosis with patent foramen ovale	Increased	Increased	Decreased	Equals vena cava	Equals right atrium	Equals right ventricle	Decreased

Clinical Findings.

A. Symptoms and Signs: Acute pericarditis with effusion produces the symptoms and signs of cardiac tamponade, which include apprehension, dyspnea, cyanosis, distended neck veins, paradoxical pulse, low systolic and low pulse pressure, increase of area of cardiac dullness, feeble or absent apex beat, and diminished heart sounds. Pericardial friction rub, liver enlargement, fever, malaise, chest pain, leukocytosis, and increased sedimentation rate may be present.

B. X-ray Findings: The chest x-ray reveals a "water-bottle"-shaped heart shadow. Pericardial effusion must be differentiated from a dilated heart.

C. Ecg. Findings: The Ecg. shows low voltage of the QRS complex.

D. Evaluation of Pericardial Fluid: The presence of effusion is confirmed by a diagnostic paracentesis (see p. 233). Bacteriologic and cytologic study of the fluid, when correlated with the clinical picture, will usually establish the nature of the underlying disease.

   1. Serofibrinous effusion is caused by collagen disease, uremia, neoplasm, allergy, and infection.

   2. Purulent fluid may develop in pyogenic and tuberculous infections. Purulent pericarditis is usually secondary to pneumonia, empyema, or septicemia.

   3. Hemorrhagic fluid is seen in tuberculosis, malignant invasion of the pericardium, anticoagulant therapy, intrapericardial rupture of aneurysm or infarct of the heart, and after trauma or surgery.

E. Pericardial Biopsy: When etiology remains uncertain, particularly if tuberculosis is suspected, open pericardial biopsy under local anesthesia should be considered.

Treatment.

A. Of Cardiac Tamponade: This must be relieved by pericardial tap (see p. 233), occasionally as an emergency measure. Fluid is withdrawn slowly to avoid acute cardiac dilatation. Air (equivalent to half the volume of fluid evacuated) may be injected into the pericardial sac for better visualization by x-ray.

B. Of Purulent Pericarditis:

   1. Systemic chemotherapeutic agents, selected by sensitivity studies and administered intensively (see p. 614).

   2. Intrapericardial antibiotics - If the organisms are sensitive to antibiotics suitable for local use (such as penicillin and streptomycin), these are injected into the pericardial sac at the conclusion of each tap.

   3. Pericardiostomy - An extrapleural pericardiostomy should be performed under local anesthesia and Penrose drains inserted into the pericardial sac if infection is not brought under control by systemic and topical antibiotics combined with evacuation of fluid by repeated taps. Enzymatic debridement by the twice daily injection of Streptokinase-Streptodornase, N.N.D. (Varidase®) (see p. 204) may improve drainage from the pericardiostomy if exudates are heavy.

CHRONIC PERICARDITIS
WITH EFFUSION AND CARDIAC TAMPONADE

Idiopathic, posttraumatic, tuberculous, and other forms of acute pericarditis occasionally progress to a subacute or chronic stage in which the pericardium is thickened and there is a persistent tendency to produce fluid. Chronic cardiac tamponade occurs in spite of repeated paracenteses, and the patient is disabled. Continued conservative management in these cases leads to prolonged invalidism and possibly to the eventual development of constrictive pericarditis. Open drainage procedures result in secondary infection of the pericardium. When this stage of chronic effusion and tamponade has been reached and there is failure of response to repeated taps and other therapy, pericardiectomy usually brings prompt relief.

In tuberculous pericarditis, when fever and signs of cardiac compression persist after a month of intensive antituberculous chemotherapy and supportive treatment, surgical excision of the pericardium may be in order. Judgment is required to determine when the disease is no longer amenable to medical management.

CHRONIC CONSTRICTIVE PERICARDITIS

In this disorder the heart is compressed by a thickened, adherent pericardium so that diastolic filling is reduced. Areas of calcification are often present in the pericardium and occasionally penetrate into the myocardium. Simple adherence of the pericardium has no significant effect; constriction must be present. The etiology of constrictive pericarditis is uncertain in over half of these patients. Tuberculous infection is considered to be the commonest single cause, and many of the so-called idiopathic cases are probably healed tuberculosis. Constrictive pericarditis occasionally follows traumatic hemopericardium and rheumatic fever.

Dyspnea, venous distention, abdominal swelling, and general weakness are the most common symptoms. Physical findings in the presence of marked cardiac compression include distant heart sounds, tachycardia, low systolic and pulse pressure, paradoxical pulse (decreased pulse on inspiration), pleural effusion, hepatomegaly, ascites, scrotal swelling, and ankle edema. Studies of the circulation show increased venous pressure, circulation time, and blood volume; the cardiac output is diminished, and catheterization of the heart reveals elevation of diastolic pressure in the right ventricle and pulmonary artery. The Ecg. has a diminished amplitude. Chest films usually show pleural effusion and may show an enlarged heart and pericardial calcification. Fluoroscopy reveals diminished cardiac pulsations. Liver function tests are often abnormal.

Constrictive pericarditis is frequently confused with primary renal or liver disease or congestive failure. The predominance of hepatomegaly and ascites and the absence of orthopnea often directs attention away from the heart and toward the liver. In making the differential diagnosis it is helpful to keep in mind Beck's diagnostic triad of constrictive pericarditis: a small, quiet heart, increased venous pressure, and ascites.

Pericardiectomy is the only satisfactory treatment. Preoperatively, removal of fluid from the chest and abdomen may be neces-

sary to improve pulmonary reserve.

If the operation is carried out in the early stages of the disease, the risk is relatively low and prompt relief of symptoms and signs can be expected. Prolonged cardiac compression causes atrophy of the heart muscle, which increases the operative risk and adversely affects the results. Early diagnosis and treatment are therefore important.

MITRAL STENOSIS

Mitral stenosis is almost always caused by rheumatic heart disease dating from childhood or adolescence. However, about half of patients with mitral stenosis are unaware of having had rheumatic fever. Congenital mitral stenosis is quite rare.

Rheumatic valvulitis produces narrowing of the mitral orifice by sealing together the free margins of the valve in a firm, fibrous union. The stricture tends to increase slowly over the years. Deposition of calcium may result in hard, irregular excrescences around the stenotic orifice, and the chordae tendineae cordis may become partially cross-fused. The normal valve opening is approximately 5 sq. cm. in area. Mild dyspnea on exertion may develop when the lumen is reduced to 2.5 sq. cm., but when the critical size of 1 sq. cm. is reached progressive symptoms invariably occur.

Significant stenosis of the mitral orifice is followed by (1) reduction of cardiac output (at first, only on exercise; later, even at rest) and (2) rise in pressure in the left atrium, pulmonary vascular bed, and right ventricle. This causes dilatation of the left atrium and pulmonary vascular bed and hypertrophy of the right ventricle. When the resting hydrostatic pressure in the left atrium and pulmonary capillaries reaches 25 mm. Hg, there is danger of rapid development of pulmonary edema on further rise (as with exercise or emotion). Pulmonary vascular changes (thickening of the arteriolar walls) take place; these are ultimately responsible for chronically increased pulmonary resistance and hypertension in the pulmonary artery and right ventricle.

Clinical Findings.

A. Symptoms and Signs: There may or may not be a history of rheumatic fever. Dyspnea on exertion and easy fatigability are the first symptoms. Cough, hemoptysis, palpitation, and paroxysmal nocturnal dyspnea are common later manifestations. The heart is usually not greatly enlarged unless other valve lesions exist. An apical rumbling diastolic murmur with presystolic accentuation is characteristic. There may be a diastolic thrill, loud apical snapping first sound, and opening snap. Atrial fibrillation is common, and when it is present there is no presystolic accentuation. The pulmonary second sound is increased, and there may be a murmur of pulmonary insufficiency. Other valve defects and their typical murmurs may be present. Venous engorgement, liver enlargement, and peripheral edema are signs of right heart failure.

B. X-ray Findings: Enlargement of the left atrium, pulmonary artery, and right ventricle are typical. The pulmonary

markings are prominent. Barium swallow shows displacement
by the enlarged left atrium.

C. Ecg. Findings: P wave abnormality and right ventricular hy-
pertrophy are the common findings. The presence of left ven-
tricular hypertrophy indicates the coexistence of another defect
such as mitral regurgitation or aortic valvular disease.

D. Cardiac Catheterization: Catheterization is not necessary for
diagnosis but is of value in determining the degree of functional
impairment. Repeat catheterization postoperatively provides
objective evidence of the success or failure of surgery. Find-
ings on catheterization are increased pulmonary capillary,
pulmonary arterial, and right ventricular pressure. Cardiac
output tends to be low, with no or limited increase on exercise.
The contour of the wedge pressure tracing may suggest (but
not prove) the presence of significant mitral regurgitation, an
important preoperative consideration. Left atrial puncture
occasionally provides additional useful data.

Treatment.

Mitral commissurotomy is of benefit in a selected group of pa-
tients. The operation usually consists of separation of the fused
cusps and chordae tendineae cordis with a finger inserted through
the left atrial appendage or the right interatrial groove. Cutting or
dilating instruments may also be required. This procedure has the
disadvantage of being carried out blindly within the heart by the
sense of touch. Direct exposure of the mitral valve with the aid of
extracorporeal circulation permits more adequate correction of the
deformity. For this reason, the open heart technic is now being
used with increasing frequency and will eventually become the method
of choice.

A. Indications for Mitral Commissurotomy: As an aid in selection,
the Standard Classification of Heart Disease of the New York
(American) Heart Association is widely used. A simplified
form of this classification is as follows: Group I: Asympto-
matic. Group II: Mild symptoms and no serious exercise in-
tolerance. Group III: Clear-cut symptoms, becoming more
severe, and precluding many accustomed activities. Group IV:
Totally incapacitated, with relatively intractable cardiac failure.
Patients in Groups II and III are suitable for operation, and
the surgical mortality should be less than 5%. Group IV pa-
tients are desperate risks but may occasionally be improved by
commissurotomy. Specifically, operation should be considered
in the presence of any of the following clinical developments:
(1) increasing exertional dyspnea, (2) onset of atrial fibrillation,
(3) peripheral embolism, (4) major hemoptysis, (5) signs of
pulmonary congestion such as paroxysmal nocturnal dyspnea,
(6) history of congestive failure, (7) severe symptoms in preg-
nancy, (8) roentgenographic evidence of pulmonary hypertension,
and (9) Ecg. evidence of right ventricular hypertrophy.

B. Contraindications to Commissurotomy: These include active
rheumatic carditis, bacterial endocarditis, a significant degree
of mitral insufficiency, intractable cardiac failure (Group IV),
and the presence of other serious disease with a poor prognosis.
Age itself is not a contraindication to operation, but patients
over 50 must be selected with care. Unfavorable factors which

tend to vitiate results but do not contraindicate surgery are
chronic atrial fibrillation, heavily calcified valves, and ad-
vanced pulmonary and myocardial damage from long-standing
mitral stenosis.

C. Preoperative Preparation: Before operation all signs of fail-
ure should be controlled by the proper use of rest, diuretics,
and salt restriction. Whether or not failure is present, every
patient must be fully digitalized preoperatively: if rhythm is
normal, quinidine should be administered for 1 day preopera-
tively and continued postoperatively as a prophylactic measure.
(Dosage: 0.2 Gm. every 6 hours to a total of 0.8 Gm. daily.)

D. Complications of Commissurotomy: In addition to acute con-
gestive failure and the usual post-thoracotomy complications,
mitral commissurotomy patients are especially subject to
peripheral embolism, atrial fibrillation, and the postcommis-
surotomy syndrome.

1. Peripheral embolism - Emboli may be dislodged at oper-
ation from a mural or atrial thrombus in the left atrium
or from a calcified valve margin. The incidence of thrombo-
embolism in all commissurotomies is variously reported as
4-16%; the incidence is at least doubled in the presence of
atrial fibrillation, atrial thrombosis, or previous embolism.
Unfortunately, about 65% of emboli are cerebral and not
surgically accessible. Maneuvers at operation such as oc-
clusion of the carotid arteries in the chest or in the neck
have not significantly reduced the incidence of cerebral em-
bolism. Embolism usually occurs during operation, but the
danger persists throughout the postoperative period.

   Preoperatively, all major pulses should be palpated and
recorded. Immediately postoperatively, while the patient
is still anesthetized, these pulses should be rechecked. Oc-
clusion of a major artery is an indication for immediate em-
bolectomy. After a patient has recovered completely from
an adequate commissurotomy, embolism is extremely rare
even though embolism may have been a problem preopera-
tively.

2. Atrial fibrillation - This arrhythmia exists preoperatively
in 30-40% of patients. In 20-40% of patients with normal
sinus rhythm preoperatively, atrial fibrillation occurs after
surgery, most commonly on the second to fourth days. The
more severe the cardiac disease, the greater the tendency
to postoperative arrhythmia. Preoperative administration
of digitalis and quinidine reduces the incidence of atrial
fibrillation significantly.

   If atrial fibrillation develops postoperatively and the pa-
tient is receiving digitalis and quinidine, reversion to sinus
rhythm can usually be accomplished by further slowing of
the cardiac rate with increased digitalis dosage or by a sub-
sequent course of quinidine. If a patient develops atrial
fibrillation postoperatively and is not receiving digitalis and
quinidine, he should be fully digitalized and, when his gen-
eral condition is satisfactory, given quinidine in an effort
to convert the rhythm to normal.

3. Postcommissurotomy syndrome - Pericardiotomy for ac-
quired or congenital heart disease may be followed by a

syndrome of precordial chest pain, fever, and sometimes cough, with or without pleurisy and pericarditis. Symptoms may begin at any time from a few days to months following the operation, persist for 1-4 weeks, and subside spontaneously. It is important to rule out other causes of these symptoms, particularly sepsis. Otherwise the condition is self-limiting and is treated symptomatically with salicylate analgesics. Reactivation of rheumatic fever by commissurotomy is rare, and is not the cause of the postcommissurotomy syndrome.

E. Results of Mitral Commissurotomy: About 70% of patients surviving an adequate commissurotomy have significant subjective improvement over their preoperative status after 5 years. Objective evidence of improvement is often not so conclusive. Poor results are usually related to inadequate commissurotomy, production of significant mitral regurgitation by the procedure, recurrence of mitral stenosis, involvement of other valves, or advanced myocardial or pulmonary damage.

MITRAL REGURGITATION

The cause of mitral regurgitation is rheumatic heart disease. The distinguishing features are diminished cardiac reserve, cardiac enlargement, left ventricular lift, apical systolic murmur (which may be accompanied by a thrill) radiating to the axilla and back, and a loud pulmonic second sound. Atrial fibrillation is common. X-ray of the chest shows enlargement of the left ventricle, left atrium (often marked), and pulmonary artery. Pulmonary congestion may be present. The left ventricle is under severe strain, and there may be signs of congestive failure. The Ecg. indicates left ventricular hypertrophy.

Slight regurgitation is often associated with mitral stenosis, the latter being the major problem. Under these circumstances, mitral commissurotomy by one of the closed technics is performed with care to avoid increasing the regurgitation. The surgical management of mitral regurgitation, when it is the primary defect, is in the stage of rapid evolution. Open operation with cardiopulmonary bypass is now the procedure of choice. Plication of the dilated annulus and plastic revision of the deformed valve is the usual objective. Replacement of the mitral valve by a prosthetic device has been tried but still must be regarded as experimental.

AORTIC STENOSIS

Aortic stenosis may be congenital, rheumatic, or arteriosclerotic. Congenital stenosis is usually discovered in children or young adults (see p. 236). Rheumatic aortic stenosis occurs primarily in young and middle-aged adults with a history of rheumatic fever; mitral stenosis is often associated, in which case the disorder tends to follow a protracted course characteristic of mitral disease. Arteriosclerotic aortic stenosis is a disease predominantly of men over the age of 50. Although aortic stenosis occurs over a wide

span of age and is of varied etiology, the clinical features and surgical implications of all types are somewhat similar.

Clinical Findings.

A. **Symptoms:** Fatigue and palpitation are common early complaints. The classic triad of angina, syncope, and left ventricular failure indicate impending disaster. Patients may die unexpectedly in an episode of fainting, in a Stokes-Adams attack, or in ventricular fibrillation.

B. **Signs:** Cardiac enlargement and forceful apical beats are usually present. The most characteristic finding is a harsh systolic murmur, often accompanied by a thrill, maximal in the left second or third interspace along the left sternal border and well transmitted to the neck vessels. The aortic second sound may be diminished. The BP may be normal or low, and the pulse pressure may be diminished.

C. **X-ray Findings:** The only suggestive changes are left ventricular enlargement and, frequently, valvular calcification, which is best demonstrated by fluoroscopy or spot films.

D. **Ecg. Findings:** A left ventricular hypertrophy pattern is usually present. When mitral stenosis is also present, there may be no evidence of ventricular hypertrophy.

E. **Cardiac Catheterization:** Combined pressure tracings from the left ventricle and a peripheral artery show left ventricular hypertension and a gradient across the aortic valve.

Treatment.

Indications for operation in the rheumatic and arteriosclerotic types of stenosis are controversial. Technics for correcting these severely deformed valves have not been standardized. Dilatation of the valve with an instrument passed through the apex of the left ventricle is the safest method, but open transaortic surgery employing hypothermia or extracorporeal circulation is more effective. Because aortic valve surgery is still in the developmental stage, these patients should be selected for operation with care. The optimal candidate is 40 years of age or under, and it is probably not wise to operate on patients over 60. The gradient across the mitral valve should be at least 50 mm. Hg (25 mm. Hg is acceptable as the lower limit in the presence of mitral stenosis). Subjects most suitable for operation are those who are showing progression of symptoms and signs on medical management and yet are still adequately controlled.

AORTIC REGURGITATION

Aortic insufficiency is usually caused by rheumatic valvulitis, but may also be due to congenital defect, subacute bacterial endocarditis, syphilis, or trauma. No symptoms are present in the early stages, but in time progressive myocardial strain causes fatigue, dyspnea, paroxysmal nocturnal dyspnea, angina, and eventual congestive failure. As in aortic stenosis, sudden death due to arrhythmia is not uncommon. The diagnosis is usually obvious from the characteristic blowing diastolic murmur heard best along the left sternal border and in the second right interspace. Multiple valve lesions are sometimes present, producing various additional murmurs. There is a water-hammer pulse with a wide pulse pressure

which may be confirmed by an arterial tracing. Other diagnostic features include a boot-shaped heart when left ventricular hypertrophy and dilatation are present and Ecg. evidence of left ventricular hypertrophy.

Surgical treatment of aortic insufficiency is still in the experimental stage. Repair of the deformed valve under direct vision using extracorporeal circulation has been done successfully in a few medical centers. At present, the indications for surgical treatment and the procedures of choice have not been established, and operative mortality is quite high. Damage to the aortic valve is often so marked that total removal of the valve and replacement with a suitable prosthesis appears to be the only solution; as yet, however, no satisfactory valve substitute has been developed

TRICUSPID STENOSIS AND REGURGITATION

Tricuspid valve stenosis or regurgitation due to rheumatic fever is relatively uncommon. It usually occurs in association with damage to other valves, especially the mitral valve   Symptoms and signs are related chiefly to increased venous pressure. Easy fatigability, distention of superficial veins, hepatomegaly, abdominal fluid, and dependent edema develop as the disorder progresses. The murmur of tricuspid stenosis resembles that of mitral stenosis (which may also be present), but is best heard over the lower sternum. In tricuspid regurgitation a systolic murmur is heard low and to the right of the sternum. Tricuspid stenosis causes enlargement only of the right atrium, but regurgitation produces right ventricular hypertrophy as well   The diagnosis can be confirmed by cardiac catheterization.

Surgical treatment of tricuspid stenosis is commissurotomy carried out as for mitral stenosis. When tricuspid and mitral stenosis coexist, both valves are opened at the same operation. Tricuspid regurgitation requires an open heart approach for adequate management.

VENTRICULAR ANEURYSM

A portion of the ventricular muscle may become attenuated and fibrotic after myocardial infarction and balloon outward to form an aneurysmal dilatation. On fluoroscopy, a paradoxical pulsating mass is seen on the border of the cardiac shadow. Congestive failure is frequently present. Surgical excision can be accomplished either by closed technic or with the use of the cardiopulmonary bypass. Postinfarction aneurysm is the commonest type, but this lesion may also be congenital or due to trauma

CORONARY ARTERY DISEASE

The surgical treatment of coronary heart disease is in the phase of experiment and development. Symptomatic improvement and possibly increased longevity have been reported following epicardial abrasion and poudrage with asbestos particles (Beck operation). The

purpose of this procedure is to stimulate the development of coronary collaterals. Coronary endarterectomy has proved feasible in selected cases, but the operative risk is great at present. There are numerous other less promising approaches to the problem of improving myocardial blood supply. Patients who are severely incapacitated by angina which is not controllable by medical measures but who have sufficient myocardial reserve to withstand operation are most suitable for surgical consideration.

CARDIAC TUMORS

Primary tumors of the heart are very rare. They are clearly a surgical problem, and open heart technics are now available for their removal in favorable cases. These neoplasms may be benign (e.g., myxoma, fibroma, lipoma, rhabdomyoma) or malignant (usually sarcomatous). Myxomatous tumors in the left atrium often project over the mitral valve and simulate mitral stenosis. They may also release tumor emboli. The clinical findings in cardiac tumors as a group tend to be bizarre and to resemble those of various valvular lesions, constrictive pericarditis, or pericardial effusion. Diagnosis depends upon a high index of suspicion and often requires refined diagnostic technics such as angiocardiography. Prognosis is excellent after excision of benign tumors.

11 . . .

Surgery of the
Breast and Soft Tissues

CARCINOMA OF THE BREAST

Carcinoma of the breast is among the most common malignant tumors and is a major cause of death in women. The peak incidence is between the ages of 40 and 50, but breast cancer occurs frequently at all ages past 30.

A predisposition to breast cancer is inherited; women with a family history of mammary carcinoma are at least twice as likely to develop the disease and tend to be affected at an earlier age. Because cystic disease of the breast (see p. 255) is believed to be associated with an increased incidence of malignancy, continuous follow-up of patients with cystic disease is indicated. The effect of marital status, parity, and nursing on the incidence of breast carcinoma is not clear. Inflammation, trauma, and benign neoplasms are not precancerous.

Although the mean duration of life in untreated carcinoma of the breast is about 3 years, the biologic behavior of the disease is highly variable; some untreated patients succumb in 3 months, whereas others survive for 5-30 years. In general, the course of mammary cancer is related to histologic type and grade: well-differentiated tumors tend to progress more slowly. However, the choice of treatment is based primarily upon the extent of the disease and not upon the microscopic pattern of the lesion.

The relative frequency of carcinoma in various anatomic sites in the breast is as follows: Upper outer quadrant, 45%; lower outer quadrant, 10%; upper inner quadrant, 15%; lower inner quadrant, 5%; central (subareolar or diffuse), 25%. Metastasis to regional lymph nodes is the principal mode of spread. Axillary metastases are found on microscopic study in 50-60% of patients undergoing radical mastectomy. The internal mammary nodes are invaded in about one-third of patients who have clinically advanced disease of borderline operability. When the tumor is in the central or inner half of the breast and when the axillary nodes have already been invaded, the internal mammary chain is particularly likely to be involved.

Hematogenous spread of breast cancer is common; the bones (especially the pelvis, spine, femur, ribs, skull, and humerus), lungs, and liver are most frequently affected.

Clinical Findings.
  A. Symptoms and Signs: The primary complaint in about 80% of patients with breast cancer is a lump (usually painless) in the breast. Less frequent symptoms are breast pain; erosion, retraction, enlargement, discharge, or itching of the nipple; and redness, generalized hardness, enlargement, or shrinkage of the breast. Rarely, an axillary mass, swelling of the arm, or back pain (from metastases) may be the first symptom.

Examination of the breast should be meticulous, methodical, and gentle. Careful inspection and palpation - with the patient supine, arms at her sides and overhead; and sitting, arms at her sides and overhead - are essential; unless this procedure is followed at all physical examinations, early lesions will be missed. In some series, 5-10% of cases of breast carcinoma have been discovered during physical examinations performed for other purposes.

A lesion smaller than 1 cm. in diameter may be difficult or impossible for the examiner to feel and yet may be discovered by the patient. She should always be asked to demonstrate the location of the mass; if the physician fails to confirm her suspicions, he should repeat the examination in 1 month. During the premenstrual phase of the cycle increased innocuous nodularity may suggest neoplasm or may obscure an underlying lesion. If there is any question regarding the nature of an abnormality under these circumstances, the patient should be asked to return after her period.

The axillary and cervical regions must be examined carefully for lymphadenopathy. The location, size, consistency, and other physical features of all lesions should be recorded on a drawing of the breast for future reference.

Breast cancer usually consists of a nontender firm or hard lump with poorly delimited margins (caused by local infiltration). Slight skin or nipple retraction is an important early sign. Minimal asymmetry of the breast may be noted. Very small (1-2 mm.) erosions of the nipple epithelium may be the only manifestation of carcinoma of the Paget type. Watery, serous, or bloody discharge is an infrequent early sign (see p. 254). The following are characteristic of advanced carcinoma: edema, redness, nodularity, or ulceration of the skin; the presence of a large primary tumor; fixation to the chest wall, enlargement, shrinkage, or retraction of the breast; marked axillary lymphadenopathy; and distant metastases.

B. Special Clinical Forms of Breast Carcinoma:

   1. Paget's carcinoma - The basic lesion is intraductal carcinoma, usually well-differentiated and multicentric in the nipple and breast ducts. The nipple epithelium is infiltrated, but gross nipple changes are often minimal and a tumor mass may not be palpable. The first symptom is often itching or burning of the nipple accompanied by a superficial erosion or ulceration. The diagnosis is readily established by biopsy of the erosion.

      Paget's carcinoma is not common (about 3% of all breast cancers), but it is important because it appears innocuous. It is frequently diagnosed and treated as dermatitis or bacterial infection. This is disastrous, for the lesion metastasizes to regional nodes in up to 60% of cases and should be treated in the same manner as other forms of breast cancer.

   2. Inflammatory carcinoma - This is the most malignant form of breast cancer and comprises about 3% of all cases. The clinical findings consist of a rapidly growing, sometimes painful mass which enlarges the breast. The overlying skin becomes erythematous, edematous, and warm. The diagnosis should be made only when the redness involves more

than one-third of the skin over the breast. The inflammatory changes, often mistaken for an infectious process, are caused by carcinomatous invasion of the subdermal lymphatics with resulting edema and hyperemia. These tumors may be caused by a variety of histologic types. Metastases occur early and widely in all cases, and for this reason inflammatory carcinoma is virtually incurable. Radical mastectomy is not advised. Radiation and hormone therapy are of little value.

C. Laboratory Findings: A consistently elevated sedimentation rate or serum alkaline phosphatase is suggestive of widespread metastases.

D. X-ray Findings: Because of the frequency of metastases to the bones and lungs, preparation for a radical mastectomy should usually include posteroanterior and lateral chest films, anteroposterior and lateral views of the lumbar spine and pelvis, and a lateral skull x-ray.

Differential Diagnosis.

Differential diagnosis depends upon biopsy. The following lesions are most likely to be confused with carcinoma: cystic disease of the breast, adenosis, adenofibroma (in the older patient), intraductal papilloma, and fat necrosis.

Treatment.

A. Surgical Treatment: All malignant lesions confined to the breast and axillary nodes should be treated by radical mastectomy if the patient's general health permits. Few patients, regardless of age, are unable to withstand a properly conducted operation.

The criteria of operability established by C. D. Haagensen in Diseases of the Breast (Saunders, 1956) are of great value in selecting patients who may benefit from surgical treatment. He advises radical mastectomy except when:

1. Extensive edema of the skin over the breast (more than one-third of the skin area) is present.
2. Satellite nodules are present in the skin over the breast.
3. The carcinoma is of the inflammatory type.
4. Any 2 or more of the following grave signs of locally advanced carcinoma are present:
    a. Ulceration of the skin.
    b. Edema of the skin of limited extent (less than one-third of the skin over the breast).
    c. Solid fixation of the tumor to the chest wall.
    d. Axillary lymph nodes measuring 2.5 cm. or more in transverse diameter.
    e. Fixation of axillary nodes to the skin or deep structures of the axilla.
5. Edema of the arm is present.
6. Palpable supraclavicular nodes are present and biopsy shows metastases.
7. Biopsy of the internal mammary nodes in the first, second, or third interspaces and/or at the apex of the axilla reveals metastases.

      a. Internal mammary biopsies are done for patients in whom (1) the primary tumor is situated in the lower parasternal zone of the breast; (2) the primary tumor measures more than 3 cm. in diameter; (3) any of the 5 grave signs of locally advanced disease (see para. 4 above) are present; (4) the axillary nodes are clinically involved.

      b. Apex of axilla biopsies are done for patients in whom (1) there is a single clinically involved axillary node measuring 2.5 cm. or more in diameter; (2) there are more than 2 clinically involved nodes; (3) the node or nodes are fixed; (4) the primary tumor measures more than 5 cm. in diameter; (5) any of the 5 grave signs of locally advanced disease (see para. 4 above) are present.

   8. Distant metastases are demonstrated by roentgenographic study of the chest, by palpation of the liver, or by roentgenographic search for metastases in the skeletal system. In patients with pain in the back or pelvic area (suggesting vertebral metastases), trephine biopsy of the lumbar vertebrae is performed if x-rays are negative.

The selection of candidates on the basis of Haagensen's criteria will limit radical mastectomy to patients for whom it may be curative. About 30% of patients chosen for internal mammary or apex of axilla biopsy on the basis of the indications outlined above will be found to have involved nodes in 1 or both of these locations. Such patients are not curable by surgery and should be spared radical mastectomy and treated by irradiation. Multiple biopsies to determine operability should be done in a separate preliminary operative session under general anesthesia.

B. Radiotherapy: The use of radiotherapy with or without simple mastectomy as the sole means of treating breast cancer is advisable only when the tumor is too advanced or the patient's condition too poor for radical mastectomy. Postoperative irradiation of the internal mammary, axillary, and supraclavicular regions may be of value in the small group of patients in whom extensive axillary metastases are found on microscopic examination of a specimen taken during radical mastectomy. Local chest wall recurrence after radical mastectomy should be treated by x-ray rather than excision. Bone metastases, if sufficiently localized, are best managed by radiotherapy. Temporary relief of bone pain is obtained in 60-70% of such cases. Local palliation of large, ulcerated, or otherwise inoperable lesions is usually most successfully achieved by irradiation.

C. Hormone Therapy: Hormone therapy is usually employed when surgery and irradiation have failed or when widespread metastases have rendered them useless. Hormone treatment does not cure, but may temporarily retard progression of the disease. The mode of action of hormones on breast cancer is not known. Therapy is of 2 types: (1) administration of 1 of the various estrogenic or androgenic hormones, or (2) removal of the ovaries, adrenals, or pituitary.

  1. Administration of hormones -

    a. Estrogen therapy - Estrogens should be reserved for older patients, both because it is unwise to give estrogens to premenopausal women and because the effects

are more beneficial in older women. In postmenopausal women estrogen produces regression of soft tissue carcinoma in about 50% of cases. Treatment usually consists of giving diethylstilbesterol, 5 mg. t.i.d. to a total dose of about 4 Gm. for maximal response. The commonest side effects are anorexia, nausea, and vomiting; these usually disappear within a few weeks, but when symptoms of toxicity are severe the dosage should be reduced temporarily until tolerance is acquired. Pigmentation of nipples, areolas, and axillary skin, engorgement of the breasts, and uterine bleeding may occur. Caution: In patients with extensive bone metastases, estrogens may precipitate hypercalcemia followed by anuria and death.

b. Androgen therapy - Androgens give the best results in premenopausal women with soft tissue metastases or in patients with bone metastases at any age. Over half of patients report subjective improvement, and regression of bone lesions is objectively observed in 20-30% of cases. Testosterone propionate is the most effective androgen preparation. The usual dose is 50-100 mg. I.M. 3 times a week. About 3 months of treatment are required for maximal effect. Methyltestosterone is also effective and may be given in buccal tablets (50-100 mg. daily) or orally (gradually increasing dosage from 0.3 Gm. daily to 1 Gm. daily). The favorable results of androgen therapy, aside from relief of pain, are a feeling of well-being and gain in weight. The principal side reactions are the masculinizing effects, e.g., hoarseness, hirsutism, loss of scalp hair, acne, and ruddy complexion.

2. Oophorectomy, adrenalectomy, and hypophysectomy - The indications for and results of these operations in metastatic breast cancer are controversial.

a. Routine oophorectomy has been recommended in premenopausal women with breast cancer in the hope of lessening the incidence of recurrence after radical mastectomy. Extensive soft tissue recurrences in premenopausal women may respond to oophorectomy, especially if this operation is combined with total adrenalectomy.

b. Adrenalectomy or hypophysectomy for advanced breast cancer is now under study. Some patients improve following the operation, presumably as a result of reduction in endogenous estrogen production. These extensive surgical procedures should not be considered until all other methods fail.

Complications of Radical Mastectomy.

Except for local recurrence, usually due to implantation of tumor cells in the wound at operation, the only important late complication of radical mastectomy is edema of the arm. Significant edema occurs in 10-30% of cases. When it appears in the early postoperative period it is usually caused by lymphatic obstruction due to infection in the axilla. Late or secondary edema of the arm may develop years after radical mastectomy as a result of infection

in the hand or arm with obliteration of lymphatic channels. After radical mastectomy the lymphatic drainage of the arm is always compromised and the extremity is more susceptible to infection from minor injuries than formerly. The patient should be warned of this and treatment instituted promptly if infection occurs. The management of well established chronic edema by elevation and elastic support is not very successful.

Prognosis.
The five-year clinical cure rate of all patients treated by radical mastectomy is 40-60%, and the local recurrence rate is about 15%. When there is no evidence of axillary or distant metastases at the time of operation, the five-year cure rate is 75-85%. Operative mortality is about 1%. The most unfavorable anatomic site for breast carcinoma is the medial portion of the inner lower quadrant. Breast cancer is probably more malignant in young than in old women, but the difference is not great. The prognosis of carcinoma of the breast occurring during lactation or pregnancy is generally poor, since over one-fourth are inoperable; but when radical mastectomy is feasible the over-all five-year clinical cure rate in this group of patients is about 30%. The presence of axillary metastases in patients who are pregnant or lactating is an extremely poor prognostic sign.

DIFFERENTIAL DIAGNOSIS OF NIPPLE DISCHARGE

In order of frequency, the following lesions produce nipple discharge: intraductal papilloma, carcinoma, cystic disease, and ectasia of the ducts. The discharge is usually serous or bloody. When papilloma or cancer is the cause, a tumor can frequently be palpated beneath or close to the areola.
The site of the duct orifice from which the fluid exudes is a guide to the location of the involved duct. Gentle pressure on the breast is made with the fingertip at successive points around the circumference of the areola. A point will often be found at which pressure produces discharge. The dilated duct or a small tumor may be palpable here. The involved area should be excised by a meticulous technic which ensures removal of the affected duct and breast tissues immediately adjacent to it. If a tumor is present it should be biopsied and a frozen section done to determine whether cancer is present. When localization is not possible and no mass is palpable, the patient should be reexamined every week for 1 month. When unilateral discharge persists, even without definite localization or tumor, exploration must be considered. The alternative is careful follow-up at intervals of 1-3 months.
Ten to 20% of patients with serous or bloody nipple discharge prove to have carcinoma. Although none of the benign lesions causing nipple discharge are precancerous, they may coexist with cancer and it is not possible to distinguish them definitely from malignancy on clinical grounds. Patients with carcinoma almost always have a palpable mass, but in rare instances a nipple discharge may be the only sign. For these reasons chronic nipple discharge is usually an indication for exploration of the breast.

ADENOFIBROMA OF THE BREAST

This common benign neoplasm occurs most frequently in young women, usually within 20 years after puberty. It is somewhat more frequent and tends to occur at an earlier age in Negro than in white women. Multiple tumors in 1 or both breasts are found in 10-15% of patients.

The typical adenofibroma is a round, firm, discrete, relatively movable, nontender mass 1-5 cm. in diameter. The tumor is usually discovered accidentally. Clinical diagnosis in young patients is generally not difficult. In women over 30, cystic disease of the breast, adenosis, and carcinoma must be considered. Treatment in all cases is excision and frozen section to determine if the lesion is cancerous.

Cystosarcoma phylloides is a type of adenofibroma with cellular stroma which tends to grow rapidly. This tumor may reach a large size, and if inadequately excised will recur locally. The lesion is rarely malignant. Treatment is usually by local excision of the mass and a margin of surrounding breast tissue.

CYSTIC DISEASE OF THE BREAST

This disorder, also known as chronic cystic mastitis, is the most frequent lesion of the breast. It is common in women 30-50 years of age but rare in postmenopausal women, which suggests that it may be related to ovarian activity. Estrogenic hormone is considered an etiologic factor. The typical pathologic change in the breast is the formation of gross and microscopic cysts from the terminal ducts and acini. Large cysts are clinically palpable and may be several cm. or more in diameter.

Clinical Findings.

Cystic disease may produce an asymptomatic lump in the breast which is discovered by accident, but pain or tenderness often calls attention to the mass. In many cases discomfort occurs or is increased during the premenstrual phase of the cycle, at which time the cysts tend to enlarge rapidly. Fluctuation in size and rapid appearance or disappearance of a breast tumor are common in cystic disease. Multiple or bilateral masses are not unusual, and many patients will give a past history of transient lump in the breast or cyclic breast pain. Pain, fluctuation in size, and multiplicity of lesions are the features most helpful in differentiation from carcinoma. However, if skin retraction is present, the diagnosis of cancer should be assumed until disproved by biopsy.

Treatment.

When cystic disease cannot be clearly distinguished from carcinoma on the basis of the clinical findings, the patient should be prepared for radical mastectomy and the lesion explored in the operating room under general anesthesia with provisions for immediate diagnosis by frozen section. Discrete cysts or small localized areas of cystic disease should be excised when cancer has been ruled out by microscopic examination. Surgery in cystic disease should be conservative, since the primary objective of surgery

is to exclude malignancy. Simple mastectomy or extensive remov-
al of breast tissue is rarely, if ever, indicated.

When the diagnosis of cystic disease has been established by
biopsy or is practically certain because the history is classical,
aspiration of a discrete mass is justifiable. The skin and overly-
ing tissues are anesthetized by infiltration with 1% procaine and a
No. 19 gauge needle is introduced. If a cyst is present, typical
watery fluid (straw-colored, gray, greenish, brown, or black) is
easily evacuated and the mass disappears. The patient is reexam-
ined at intervals of 2-4 weeks for 3 months and every 6 months
thereafter throughout life. If no fluid is obtained, if a mass per-
sists after aspiration, or if at any time during follow-up an atypical,
persistent lump is noted, biopsy should be performed without delay.

Breast pain associated with generalized cystic disease is best
treated by avoidance of trauma and by wearing (night and day) a
brassiere which gives good support and protection. Hormone ther-
apy is not advisable because it does not cure the condition and has
undesirable side-effects.

Prognosis.

Exacerbations of pain, tenderness, and cyst formation may
occur at any time until the menopause, when the symptoms of
cystic disease subside. The patient should be taught to examine
her own breasts each month just after menstruation and to inform
her physician if a mass appears.

FAT NECROSIS

Fat necrosis is a rare lesion of the breast but is of clinical
importance because it produces a mass, often accompanied by skin
or nipple retraction, which is indistinguishable from carcinoma.
Trauma is presumed to be the cause, although only about half of
patients give a history of injury to the breast. Ecchymosis is oc-
casionally seen near the tumor. Tenderness may or may not be
present. If untreated, the mass associated with fat necrosis grad-
ually disappears. As a rule the safest course is to obtain a biopsy.
When carcinoma has been ruled out, the area of involvement
should be excised.

BREAST ABSCESS

During nursing an area of redness, tenderness, and induration
not infrequently develops in the breast. In the early stages the in-
fection can often be reversed by discontinuing nursing with that
breast and administering a broad-spectrum antibiotic. If the lesion
progresses to form a localized mass with increasing local and sys-
temic signs of infection, an abscess is present and should be
drained.

A subareolar abscess may develop in young or middle-aged
women who are not lactating. These infections tend to recur after
incision and drainage unless the area is explored in a quiescent in-
terval with excision of the involved collecting ducts at the base of
the nipple.

Except for the subareolar type of abscess, infection in the breast is very rare unless the patient is lactating. Therefore, findings suggestive of abscess in the nonlactating breast require incision and biopsy of any indurated tissue.

CARCINOMA OF THE MALE BREAST

Breast cancer is rare in men. The average age of occurrence is about 60; cases below the age of 30 are exceptionally rare. A painless lump, occasionally associated with nipple discharge, retraction, erosion, or ulceration, is the chief complaint. Examination usually shows a hard, ill-defined, nontender mass beneath the nipple or areola.

Treatment consists of radical mastectomy in operable patients, who should be selected according to the same criteria as for female breast carcinoma. The absolute five-year survival of men with breast carcinoma is poor (about 30%). Five-year survival after radical mastectomy is about 40%. Radiation therapy is employed for metastatic disease with the same indications as in women. Estrogen therapy occasionally causes regression or temporary arrest, but the most beneficial form of hormone treatment in disseminated cancer of the male breast is orchiectomy. When the effects of orchiectomy have dissipated, adrenalectomy or hypophysectomy is reported to be of value.

GYNECOMASTIA

Hypertrophy of the male breast may result from a variety of causes. Puberal hypertrophy is very common during adolescence and is characterized by a tender discoid enlargement 2-3 cm. in diameter beneath the areola with hypertrophy of the breast. The changes are usually bilateral and subside spontaneously within a year in the majority of cases.

Men between 50 and 70 will occasionally develop hypertrophy (often unilateral) similar to that occurring at puberty.

Certain organic diseases may be associated with gynecomastia: cirrhosis of the liver, hyperthyroidism, Addison's disease, testicular tumors (especially chorio-epithelioma), and adrenal cortical tumors.

If there is uncertainty about the diagnosis of the breast lesion, a biopsy should be done to rule out cancer. Otherwise, the treatment of gynecomastia is nonsurgical unless the patient insists on excision for cosmetic reasons. In this case at least 2 years should be allowed for possible subsidence.

SARCOMA OF THE SOFT TISSUES

Sarcoma of the soft tissues, although rare, is of special importance because adequate initial management will significantly reduce the local recurrence rate. The most prevalent histologic types (in approximate order of incidence) are fibrosarcoma, liposarcoma, rhabdomyosarcoma, myxoma, synovioma, Kaposi's sarcoma, and

malignant neurilemmoma. Histogenesis may be difficult to determine, which accounts for the high incidence of "fibrosarcoma," a term applied loosely to many neoplasms for want of a better designation. Sarcoma of the skin (melanoma), lymph nodes (lymphoma), and smooth muscle (leiomyosarcoma) is not included in this discussion.

Soft tissue sarcoma involves the extremities, especially the legs, more frequently than the trunk. All age groups are affected, but the highest incidence is between the ages of 30 and 60. In general, these lesions extend locally beyond their visible and palpable margins, tend to metastasize via the blood stream to the lungs, and are radioresistant. The rate of growth and spread is quite variable.

Principles of Therapy.
   A. The diagnosis of soft tissue masses should be clearly established without delay by excision. Whenever tissue is removed, microscopic study should be carried out even though the gross appearance is benign.
   B. The treatment of soft tissue sarcoma is best planned after the histologic diagnosis is established.
   C. The choice of definitive operative procedure will depend upon the anatomic location of the sarcoma. Enucleation or local excision with narrow margins is inadequate and dangerous. Subcutaneous tumors require wide removal of surrounding tissues, including skin and underlying fascia. A split-thickness graft is frequently needed. In deeper lesions, the entire muscle bundles surrounding the neoplasm should usually be removed at points of origin and insertion. During the removal of the sarcoma, the tumor itself must never be exposed, and the site of biopsy or excision should be excised with the tumor. When adequate ablation of the sarcoma is otherwise impossible, amputation is necessary.
   D. Radiation therapy is reserved for cases unsuitable for surgery.

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Abdominal Surgery

PRINCIPLES OF DIAGNOSIS OF THE ACUTE ABDOMEN

Significance of Acute Abdominal Pain.

Acute, severe abdominal pain must be assumed to be due to a surgical disorder until a definitive diagnosis has been made. Such pain is more likely to be of surgical significance when it occurs in a previously well patient, lasts more than 6 hours, or when signs of peritoneal irritation or shock are present.

Control of Pain.

Narcotics may obscure signs and symptoms of diagnostic value, especially those of peritoneal irritation. If analgesia is essential before the diagnosis can be made, use minimal effective doses and allow the effects to subside at intervals to permit evaluation.

Evaluation of the Patient.

A complete history and physical examination should be done on every patient with an acute abdomen. Special attention should be paid to the mode of onset, localization, and radiation of the pain. The point of maximum tenderness often suggests the diagnosis. It is well to keep in mind, however, that patients who are elderly, acutely ill, or in shock often have less pain, tenderness, and abdominal rigidity than one might expect, and that in obese individuals tenderness and rigidity may be poorly localized. Anorexia, nausea, vomiting, and bowel irregularity are important symptoms.

Laboratory studies which should be done immediately on all patients with an acute abdomen are as follows: (1) Hgb., WBC, and differential count, (2) urinalysis, and (3) stool test for occult blood on a specimen obtained from the rectal glove. Additional examinations are selected as required for differential diagnosis.

When the diagnosis is in doubt, it is advisable to obtain an upright posteroanterior chest film and upright and supine abdominal films. If free air or intestinal obstruction is suspected and the patient is unable to stand, obtain an abdominal film with the patient lying on his side. The upright and lateral decubitus positions should be maintained for about 3 minutes before the x-ray is taken so that free air may collect in the uppermost portions of the abdominal cavity.

Diagnostic abdominal tap is a relatively safe procedure which may be of value in difficult diagnostic problems, particularly after abdominal trauma when there is no clear-cut indication for laparotomy but hemorrhage or perforation of a hollow viscus has not been ruled out. Useful information can be obtained in 50-90% of cases. Both false-negative and false-positive taps occur occasionally. Abdominal distention is not necessarily a contraindication to

the procedure. **Technic:** The abdominal wall is infiltrated with 1% procaine, and an 18 gauge, short-bevel spinal needle is carefully introduced into the abdominal cavity. The stylus of the needle is left in place until the peritoneum is entered. The stylus is then removed and the position of the needle repeatedly adjusted as gentle efforts are made to aspirate abdominal fluid. If necessary, a tap can be made in each quadrant of the abdomen. As a rule only a few ml. of fluid will be obtained even though a considerable quantity may be present within the abdomen. The fluid is examined grossly and microscopically. Gram's and Wright's stains are made as indicated. Amylase determination or culture may be advisable.

ACUTE PERITONITIS

Localized or generalized peritonitis is the most important complication of a wide variety of acute abdominal disorders. Peritonitis may be caused by infection or chemical irritation. Perforation or necrosis of the gastrointestinal tract is the usual source of infection. Chemical peritonitis occurs in acute pancreatitis and in the early stages of gastroduodenal perforation. Regardless of the etiology, certain typical features are usually present.

Clinical Findings.
A. Systemic Reaction: Malaise, prostration, nausea, vomiting, septic fever, leukocytosis, and electrolyte imbalance are usually seen in proportion to the severity of the process. If infection is not controlled, toxemia is progressive and toxic shock (see p. 11) may develop terminally.
B. Abdominal Signs:
  1. Pain and tenderness - Depending upon the extent of involvement, pain and tenderness may be localized or generalized. Abdominal pain on coughing, rebound tenderness referred to the area of peritonitis, and tenderness to light percussion over the inflamed peritoneum are characteristic. Pelvic peritonitis is associated with rectal and vaginal tenderness.
  2. Muscle rigidity - The muscles overlying the area of inflammation usually become spastic. When peritonitis is generalized (e.g., after perforation of a peptic ulcer), marked rigidity of the entire abdominal wall may develop immediately. Rigidity is frequently diminished or absent in the late stages of peritonitis, in severe toxemia, and when the abdominal wall is weak, flabby, or obese.
  3. Paralytic ileus - Intestinal motility is markedly inhibited by peritoneal inflammation. Diminished to absent peristalsis and progressive abdominal distention are the cardinal signs. Vomiting occurs as a result of pooling of gastrointestinal secretions and gas, 70% of which is swallowed air (see p. 274.)

C. X-ray Findings: Abdominal films (see p. 259) show gas and fluid collections in both large and small bowel, usually with generalized rather than localized dilatation. The bowel walls, when thrown into relief by the gas patterns, may appear to be thickened, indicating the presence of edema or peritoneal fluid.

D. Diagnostic Abdominal Tap: Occasionally useful (see p. 259).

Treatment.

The measures employed in peritonitis as outlined below are generally applicable as supportive therapy in most acute abdominal disorders. The objectives are (1) to control infection, (2) to minimize the effects of paralytic ileus, and (3) to correct fluid, electrolyte, and nutritional disorders.

A. Specific Measures: Operative procedures to close perforations, to remove sources of infection such as gangrenous bowel or an inflamed appendix, or to drain abscesses are frequently required. The cause of the peritonitis should always be identified and treated promptly.

B. General Measures: No matter what specific operative procedures are employed, their ultimate success will often depend upon the care with which the following general measures are carried out.

  1. Bed rest in the medium Fowler (semi-sitting) position is preferred.

  2. Nasogastric suction (see p. 268) is started as soon as peritonitis is suspected. It is important to prevent gastrointestinal distention by the prompt institution of suction, which is continued until peristaltic activity returns and deflation by rectum seems imminent or has begun. The gastric (e.g., Levin) tube is usually adequate. In persistent paralytic ileus, the intestinal tract may be more adequately decompressed by means of a long intestinal tube (e.g., Miller-Abbott), although passage of such a tube into the small bowel is frequently difficult because of poor intestinal motility. In rare cases combined gastric and long intestinal tube suction may be necessary to relieve or prevent distention.

  3. Give nothing by mouth. Oral intake can be resumed slowly after nasogastric suction is discontinued.

  4. Fluid and electrolyte therapy and parenteral feeding (see p. 99).

  5. Narcotics (see p. 605) and sedatives (see p. 607) should be used liberally to ensure comfort and rest.

  6. Antibiotic therapy (see p. 614) - If infection with mixed intestinal flora is probably present, combined therapy with penicillin and streptomycin is begun empirically. It is often advisable to add a third antibiotic (e.g., tetracycline or chloramphenicol) to this regimen. When cultures are available, antibiotics are chosen according to sensitivity studies.

  7. Blood transfusions are used as needed to control anemia.

  8. Toxic shock, if it develops, requires intensive treatment (see p. 11).

Complications and Prognosis.

The most frequent sequel of peritonitis is abscess formation in the pelvis (see p. 278), in the subphrenic space (see p. 57),

between the leaves of the mesentery, or elsewhere in the abdomen. Antibiotic therapy may mask or delay the appearance of localizing signs of abscess. When fever, leukocytosis, toxemia, or ileus fails to respond to the general measures for peritonitis, a collection of pus should be suspected. This will usually require surgical drainage. Abscess within the liver (see p. 326) and pylephlebitis (see p. 278) are rare complications. Adhesions may cause early or, more frequently, late intestinal obstruction.

If the cause of peritonitis can be corrected, the infection, accompanying ileus, and metabolic derangement can usually be managed successfully.

ABDOMINAL INJURIES

There are 2 general types of abdominal trauma: nonpenetrating and penetrating. The major complications which require surgical intervention are internal bleeding and perforation of a hollow viscus.

NONPENETRATING INJURIES

Clinical Findings.

Blunt injuries are difficult diagnostic problems for the following reasons: (1) They are frequently associated with other serious trauma such as multiple fractures and head injuries with coma. (2) Relatively trivial blows to the abdomen may rupture the bowel or spleen. (3) The absence of an external wound on the abdomen is deceptive. On the other hand, a severe abdominal blow may produce temporary collapse (primary or neurogenic shock), transient paralytic ileus, and a variable degree of abdominal soreness even in the absence of significant visceral damage. There is often a delay in onset of signs when a serious injury exists. For example, evidence of peritonitis may not appear for 8-12 hours or more after rupture of a hollow viscus, and slow or recurrent bleeding may not produce shock until some hours after an injury.

A. Signs of a Ruptured Hollow Viscus: When signs of localized or generalized peritonitis develop within 24 hours following blunt abdominal trauma, perforation of a hollow viscus should be suspected. The usual clinical picture consists of increasing abdominal pain, tenderness, rebound tenderness, muscle spasm, and depression of bowel sounds after a latent period of a few hours following the injury. Tachycardia, fever, and leukocytosis are usually noted. Diagnostic x-rays should be taken as soon after trauma as feasible. Pneumoperitoneum is present in about half of patients with rupture of any portion of the gastrointestinal tract. Diagnostic abdominal tap (see p. 259) may be helpful. Rupture of the duodenum or bladder is a particularly deceptive injury.

   1. Rupture of the duodenum - Retroperitoneal rupture of the duodenum is an insidious and often fatal injury. Signs of generalized peritonitis develop slowly or not at all. Deep

tenderness over the duodenum is the most constant finding. Retroperitoneal air may occasionally be found on x-ray. Fever and leukocytosis occur. The patient may not seem ill for a day or so until fulminating infection becomes manifest. A high index of suspicion is essential in the early diagnosis of this lesion. The serum amylase level may be elevated. Traumatic pancreatitis may be an associated injury.

2. Rupture of the bladder - See p. 389.

B. Signs of Ruptured Solid Viscus: The organs most frequently damaged are the kidneys, spleen, and liver, in that order. Hemorrhage is the most important complication.

1. Rupture of the kidney - See p. 387.

2. Rupture of the spleen - This is the most common indication for operation after blunt abdominal trauma. Even small tears of the spleen tend to cause continuous or recurrent bleeding. Minor accidents may cause splenic rupture. This is particularly true in children because of the pliability of the overlying rib cage. In adults, splenic injury should always be suspected when there are fractures of the lower ribs on the left side In certain diseases (e.g., malaria, typhoid, sarcoidosis, and infectious mononucleosis), the spleen is friable and ruptures on minimal trauma or may rarely rupture spontaneously.

The signs of rupture of the spleen are pain and tenderness in the left upper quadrant, left shoulder pain caused by diaphragmatic irritation (in 75% of cases), and evidence of blood loss. Shock often develops soon after injury, and sufficient blood may collect in the abdomen to cause shifting dullness. Mild generalized peritoneal irritation appears, accompanied by paralytic ileus. The Hct. and Hgb. fall as hemodilution occurs. Moderate leukocytosis is usually present, but the temperature is normal. Diagnostic abdominal tap recovers fresh blood in about 75% of cases, but a negative tap does not rule out intra-abdominal hemorrhage (see p. 259). X-rays may occasionally be of value by showing 1 or more of the following: (1) diffuse haziness in the left upper quadrant, with absent splenic shadow; (2) downward and medial displacement of the stomach and the splenic flexure; (3) elevation and limitation of the left diaphragm; (4) absent left renal and psoas shadows.

Subcapsular or retroperitoneal rupture of the spleen may occur without bleeding into the free peritoneal cavity and without generalized peritoneal irritation. Pain and tenderness in the left upper quadrant and unexplained anemia may be the only findings. X-rays may show some of the changes mentioned above.

Delayed rupture of the spleen occurs in 1 out of 6 cases, with massive hemorrhage beginning usually within 30 days after trauma. The signs are the same as for immediate rupture, but the diagnosis may be missed if a history of trauma is not elicited.

3. Rupture of the liver - More force is required to rupture the liver than the spleen, and bleeding from the liver has a greater tendency to cease spontaneously. The right lobe is

usually involved, and other injuries, such as fractures of overlying ribs and rupture or laceration of the right kidney, are frequently present. The usual signs are pain (accentuated by deep breathing), tenderness, and muscle rigidity in the right upper quadrant, and pain on top of the right shoulder referred from the diaphragm. When significant hemorrhage has occurred, generalized mild peritoneal irritation and shifting dullness due to free blood may be noted. Shock is often present. The Hct. and Hgb. are low and the WBC is elevated. An abdominal tap may be of value (see p. 259).

Treatment.
A. General Measures: A period of observation may be necessary before a diagnosis of serious intra-abdominal injury can be made. Some or all of the following measures are advisable during this interval.
   1. Place the patient at bed rest, record the vital signs (BP, pulse, and respiration) regularly, and observe for increasing abdominal findings or for deterioration of vital signs. Administer narcotics cautiously.
   2. Give nothing by mouth and administer parenteral fluids as required (see p. 99). Nasogastric suction is often indicated. Record the intake and output.
   3. Repeat the WBC p. r. n.
   4. Prepare blood for transfusion if internal bleeding is suspected or if operation is imminent.
   5. If bleeding is suspected, repeat the Hct. every 1-2 hours. Signs of actual or impending shock are the most reliable evidence of hemorrhage. If these signs are equivocal and there is a question of significant, continuing blood loss, administer 500 ml. of blood rapidly. If response is prompt and sustained, bleeding is not massive. Failure to respond to two 500 ml. transfusions, or a relapse into shock when they have been completed, indicates continuing major blood loss or fulminating peritonitis due to gastrointestinal perforation. In either case, immediate operation is required.
B. Surgical Treatment: Rupture of a hollow viscus and internal bleeding are absolute indications for laparotomy.

PENETRATING ABDOMINAL WOUNDS

Perforation of a Hollow Viscus.
   Any knife, projectile, or other type of wound which could involve the free peritoneal cavity should be assumed to have lacerated a hollow viscus. Immediate operation is indicated in these cases. It is unwise to await the signs of peritonitis, which are often delayed for many hours since leakage may not occur at once. Probing of the wound is not a reliable means of determining the extent of injury.
   All patients with a perforated hollow viscus should be treated for acute peritonitis (see p. 260). Thoracoabdominal wounds are discussed on p. 206. Injuries of the colon and rectum deserve special comment because they produce the most rapid and serious peritoneal infection. In selected instances lacerations of the colon may be closed primarily, but extensive wounds require resection

and proximal colostomy or exteriorization. Penetrating wounds of the buttocks or pelvis frequently perforate the rectum; blood is found on rectal and proctoscopic examination. Such injuries require early and adequate local drainage and sigmoid colostomy.

Laceration of a Solid Viscus.
Intra-abdominal hemorrhage is a common, serious complication of penetrating wounds. The liver, spleen, or kidneys may be torn, or mesenteric or other large abdominal vessels may be cut. Management is as for nonpenetrating trauma with bleeding (see p. 264).

ACUTE MECHANICAL OBSTRUCTION*

The symptoms of mechanical obstruction of small or large bowel are similar, but these entities must be distinguished because their treatment differs. It is always of primary importance to determine whether strangulation (necrosis) of bowel has occurred, since this is an indication for emergency operation to prevent bowel rupture and peritonitis.

Etiology.
A. Small Bowel Obstruction: Approximately one-third of cases are due to incarceration or strangulation of an external hernia. For this reason, the various hernia sites should always be carefully palpated. Adhesions and bands (inflammatory, neoplastic, or congenital) also account for about one-third of cases. Volvulus, intussusception, internal hernia, obturation (e.g., gallstone ileus), and inflammatory or neoplastic stricture of the bowel are less frequent causes of small intestinal obstruction.

B. Large Bowel Obstruction: Neoplasm is the most frequent cause. Volvulus of the sigmoid or cecum is also important, but less common. If strangulated hernia is excluded, cancer of the large bowel is the commonest cause of intestinal obstruction after middle age.

Clinical Findings.
A. Symptoms:
1. Abdominal pain - Crampy abdominal pain is typical. It is usually mid-abdominal in small bowel obstruction and hypogastric in large bowel lesions. Borborygmus may be loud and coincident with cramps. Continuous abdominal pain, when present, is suggestive of strangulation or peritonitis.
2. Obstipation - Inability to pass stools or more than small amounts of flatus is always noted. In complete obstruction neither feces nor gas will be passed by rectum.
3. Distention - Distention depends upon the accumulation of gas and intestinal secretions proximal to the lesion. In high

*Obstruction in infants and children is discussed in Chapter 7, Pediatric Surgery.

jejunal obstruction, distention may be minimal or absent.

4. Vomiting - This is usually present from the outset in small bowel obstruction. The higher the obstruction, the more marked the vomiting. In prolonged obstructions at lower levels in the small bowel, fecal vomitus may occur and is pathognomonic. Vomiting is less common and occurs later in large bowel obstruction.

B. Signs: A distended, tympanitic abdomen with hyperactive, rushing peristalsis is typical. Colicky pain coincident with rushes of peristalsis is characteristic when it occurs. Visible peristalsis on the abdominal wall is occasionally present. Late in the course of obstruction or when peritonitis has supervened, peristalsis may be very sluggish or absent. Abdominal tenderness is usually not marked and may be absent in uncomplicated obstruction. Localized tenderness usually develops over a gangrenous segment of bowel. Signs of localized or generalized peritonitis (see p. 260) suggest gangrene or rupture of the bowel wall. Fresh blood may be found in the rectum in intussusception and in carcinoma of the rectum or colon. A sigmoidoscopy is part of the physical examination in every case of colon obstruction. Obstructed patients usually have little or no fever until strangulation or peritonitis occurs.

C. Laboratory Findings:

1. Urinalysis - High sp. gr. and ketonuria, indicating dehydration and metabolic acidosis, are the only common findings.

2. Hct. - Hemoconcentration, caused by dehydration, frequently results in increase in Hct. and Hgb.

3. WBC - Normal or slight elevation is common. A high WBC suggests strangulation obstruction, but gangrenous bowel can be present in the absence of significant leukocytosis.

4. Blood chemistry - Serum sodium, potassium, chloride, and CO_2, and BUN are often deranged and should be determined as an aid to management of fluid imbalance if important losses have occurred (see p. 99).

D. X-ray Findings: Plain abdominal films in the supine and standing positions should be obtained immediately and repeated daily, or oftener if necessary, to follow the course of the illness. A film should be taken in the lateral decubitus position if the patient cannot stand. Normally, the stomach and colon contain a small amount of gas but the small bowel does not visualize. Complete small bowel obstruction is characterized by dilated small bowel, usually with fluid levels. Little or no gas is seen in the colon. In colonic obstruction the large bowel is dilated proximal to the block and may become markedly distended. When the ileocecal valve is incompetent, the small bowel is also dilated.

It is often impossible to differentiate between colonic and small bowel obstruction without a barium enema. A barium enema should be done early in most cases of large bowel obstruction. Care must be taken not to force a large volume of barium past an obstructing lesion. Barium should not be given by mouth in any patient suspected of intestinal obstruction because of the danger of inspissation of barium and aggravation of the obstruction.

Differential Diagnosis.

A. Simple vs. Strangulation Obstruction: This is the most important differential diagnosis to be made since, if strangulation has occurred, emergency operation is required. If the obstruction is simple (i.e., if necrosis of bowel is not present), conservative management is, for the moment, safe. In simple obstruction, the obstruction is often incomplete, so that the patient continues to pass some flatus.

In small bowel involvement, if the obstruction is complete (as indicated by the persistent lack of flatus or stools and by the absence of gas in the colon), it is wise to assume that strangulation may be present. Abdominal tenderness, signs of peritonitis, tachycardia, fever, and marked or rising leukocytosis are also suggestive of strangulation. Additional signs frequently associated with strangulation are (1) the presence of an abdominal, pelvic, or rectal mass; (2) a single, markedly dilated loop on x-ray; (3) blood in the rectum; and (4) the presence of constant, severe abdominal pain. It is important to realize, however, that necrosis may be present in the absence of all definitive signs.

B. Small vs. Large Bowel Obstruction: X-rays are required to make the differential diagnosis, but certain distinctive clinical features are usually present also. Large bowel obstruction is frequently relatively slow in onset and benign in course, without vomiting in spite of considerable distention. Severe cramps, frequent vomiting, and early fluid imbalance are more common in small bowel lesions. A history of previous abdominal surgery or of previous attacks of obstruction favors the diagnosis of small bowel involvement. Elderly patients without such a past history who become obstructed usually have a carcinoma of the large bowel.

C. Mechanical Obstruction vs. Paralytic Ileus: The distinguishing features of paralytic ileus are as follows: (1) There is usually an obvious exciting cause, such as abdominal surgery, acute peritonitis, or trauma to the abdomen or back; (2) the abdomen is silent, and cramps are absent; (3) tenderness may or may not be present, depending upon the underlying cause; (4) on x-ray, gas is distributed throughout the small and large intestines.

The differentiation of paralytic from mechanical obstruction is most difficult in the period immediately following abdominal surgery, when paralytic ileus may be associated with mechanical obstruction caused by adhesions. Under these circumstances, the persistence of abdominal distention, vomiting, and obstipation beyond the expected period of ileus raises the suspicion of a mechanical block and calls for investigation by x-ray and other means.

Treatment.

A. General Measures: Fluid balance must be restored and maintained by the parenteral route. Every patient should be promptly intubated with either a gastric (Levin) or a long intestinal tube and placed on constant suction.

B. Conservative Treatment: This consists of an effort to decompress the bowel and relieve the obstruction by means of

intubation with a gastric or long intestinal tube. A trial of in-
tubation as definitive treatment is warranted only when there
is no evidence of strangulation. It is most likely to be success-
ful in partial small bowel obstruction caused by adhesions. Con-
servative management must be vigilant, and immediate surgery
is indicated if evidence of strangulation appears or if the pa-
tient fails to improve. In general, definitive treatment by in-
tubation should not be attempted in complete large or small
bowel obstruction. The 2 types of tubes in common use are
the gastric (Levin) and the long intestinal.

1. Use of the gastric tube - Gastric tube suction is specifically
   indicated in pyloric and duodenal obstruction. Mild degrees
   of small or large bowel obstruction frequently respond to
   gastric suction. It may also be used pre- and postoperative-
   ly when obstruction is to be treated immediately by opera-
   tion. A No. 16 tube is passed through the nose into the
   stomach, placed on constant suction, and irrigated every
   1-3 hours with 30 ml. of saline to ensure patency.

2. Use of the long intestinal tube - Partial small bowel obstruc-
   tion is the primary indication for the long intestinal tube.
   In partial large bowel obstruction (e.g., due to neoplasm),
   decompression of the intestinal tract by the long tube may
   occasionally permit preparation of the colon for a one-stage
   resection of the obstructing lesion.

   The original long intestinal tube is the Miller-Abbott
   tube, which has a double lumen, 1 for suction and 1 for in-
   flation of the balloon after it passes the pylorus in order to
   encourage its more rapid propulsion by the peristaltic action
   of the small bowel. The Harris and Cantor tubes are 2 of
   the numerous modifications of the Miller-Abbott. They have
   a single, larger lumen for suction and are thus simpler and
   somewhat more efficient.

   The most difficult aspect of intestinal intubation is pas-
   sage of the tube through the pylorus. Four to 6 ml. of mer-
   cury are placed in the balloon of long intestinal tubes to aid
   in positioning within the stomach and also to serve as a
   bolus when the tube reaches the small bowel. A long, re-
   movable metal stylet extending to the tip of the tube can be
   used to guide the tube through the pylorus. This is often
   rapidly effective, but the method is more complicated and
   there is a risk of perforation.

   Technic of intubation:

   a. The well-lubricated tube is passed through the nostril into
      the stomach, and the gastric contents aspirated.
   b. Under fluoroscopic guidance the tip of the tube is passed
      through the pylorus and into the upper jejunum. This may
      be difficult or impossible in the obstructed patient. If
      attempts at passage under fluoroscopy fail or if fluoros-
      copy is not advisable, elevate the foot of the bed 30 cm.
      (12 in.) and place the patient on his right side, almost
      face down, with the tube about 75 cm. (30 in.) past the
      nares and on suction. With the patient in this position,
      the tube will often pass the pylorus within a few hours.
      When bile is aspirated, the tube is probably in the duo-
      denum.

c. When the tip of the tube is in the duodenum, advance the tube 20-25 cm. and place the patient in Fowler's position for 1 hour to facilitate passage beyond the ligament of Treitz.

d. After the tube reaches the jejunum, advance it 10 cm. (4 in.) an hour. The distal ileum is reached after insertion of about 1.5 meters (5 ft.) of tube if no obstruction is met.

e. The tube is kept on constant suction and irrigated with tap water or saline every hour to keep it open. The progress and effect of the tube is followed by an abdominal film daily or p.r.n.

f. Occasionally the stomach becomes distended when the long tube is in the small bowel. Under these circumstances a gastric tube is passed.

Signs of improvement after intubation are subsidence of pain and distention and decrease in the volume of suction drainage. X-ray shows less dilatation of intestinal loops, and gas can be seen in the colon. The passage of gas and then feces by rectum indicates that the obstruction is relieved.

When improvement has been observed, replace continuous suction by intermittent suction (e.g., 2 hours on and 2 hours off) for 12-24 hours and then gravity drainage for 24 hours while fluids are permitted by mouth. If oral fluids are well tolerated and good bowel function continues, the tube can be removed.

Failure to tolerate oral intake is an indication for resuming suction or for operation.

C. Surgical Treatment: Operation is indicated when strangulation obstruction is present (see p. 267) or when a lesion commonly associated with bowel necrosis is present (hernia, volvulus, intussusception in older children and adults, obturation, and complete obstruction caused by chronic adhesions). Preoperative intubation (see above) and appropriate fluid and electrolyte therapy are always advisable. Failure of the partially obstructed patient to respond adequately to decompression by intubation is an indication for surgical intervention; the partial relief which is usually provided by the tube should not lead to a false sense of security.

Prognosis.

The over-all mortality for mechanical obstruction is about 20%. Delay in surgical treatment and the presence of strangulation lead to a marked increase in morbidity and mortality.

OBSTRUCTION DUE TO EXTERNAL HERNIA

The diagnosis of strangulated hernia (see p. 267) should be considered in every case of intestinal obstruction. The various anatomic sites of hernia and abdominal incisions should be carefully palpated. Femoral hernias are frequently overlooked because they are small and because the physician fails to examine the femoral region. Hernias are often obscured in obese individuals. An irreducible, tender, tense hernia should be assumed to be strangulated,

and immediate operation is indicated. Forceful attempts to reduce such a hernia may push a gangrenous loop of bowel into the peritoneal cavity. An attempt to reduce a hernia by gentle manipulation is justifiable if incarceration is recent and painless.

OBSTRUCTION DUE TO VOLVULUS

A volvulus is a twist of a viscus on its mesentery of such a degree as to partially or completely occlude its blood supply. It is most likely to occur where the mesentery of the gastrointestinal tract is long and mobile.

Volvulus of the Stomach.
Volvulus of the stomach is rare and is usually associated with a large para-esophageal hiatus hernia. The stomach may rotate around either its longitudinal or its transverse axis. The major symptoms are severe epigastric or chest pain and vomiting.

Operation is required to reduce the volvulus and repair a hernia if present. It is necessary either to fix the stomach in position or to perform a sleeve resection in order to prevent recurrence.

Volvulus of the Small Intestine.
Volvulus of the midgut due to congenital failure of attachment of the small bowel mesentery occurs in infancy (see p. 151) and, rarely, in adult life. As a rule small bowel volvulus in adults is secondary to rotation of a loop of bowel around a fixed point such as an adhesion. This is a particularly lethal form of obstruction, since strangulation commonly occurs. Severe and persistent abdominal pain is often present.

Volvulus of the Cecum.
In 10-15% of individuals the cecum is poorly attached to the abdominal wall and is able to rotate on the ascending colon. Cecal volvulus occurs at all ages, but is most common in young adults.

Recurrent attacks may simulate appendicitis. The attack may be acute in onset, or several days of dull pain may precede it. The signs are those of intestinal obstruction. X-rays are diagnostic. The plain film will usually show a large gas bubble in the cecum in the left upper quadrant which may be mistaken for the stomach. The final differentiation is made by immediate barium enema.

Immediate operation is indicated for detorsion and fixation of the cecum or for primary resection and anastomosis. The latter is required if gangrene has occurred.

Volvulus of the Sigmoid Colon.
Sigmoid volvulus is common because the sigmoid is often redundant or may be enlarged by congenital or acquired megacolon. The clinical findings are those of large bowel obstruction. There is frequently a history of previous attacks relieved by enemas. Dilated loops of colon containing gas and fluid are seen on x-ray.

Barium enema confirms the diagnosis by outlining the obstructing twist in the sigmoid. Avoid filling the dilated sigmoid with barium, as this will aggravate the obstruction.

Patients with sigmoid volvulus are often old and debilitated, and the method of treatment must be chosen accordingly. It is frequently possible to decompress the sigmoid with a rectal tube. A sigmoidoscope is inserted to the point of torsion and, if the mucous membrane is of good color, a well-lubricated rectal tube - about 60 cm. (2 ft.) long and the diameter of a finger - is gently insinuated past the obstruction. Deflation occurs dramatically. The rectal tube is left in place several days and irrigated as necessary to keep it patent. Emergency operation is indicated when decompression is unsuccessful or when strangulation is a possibility. One of 2 types of operation is usually done: (1) simple detorsion of the sigmoid, with drainage of the dilated loop by a rectal tube passed up through the anal canal; or (2) resection of the sigmoid.

When nonoperative decompression by rectal tube is successful in a patient who is a good operative risk, it is possible to prepare the bowel and proceed in a few days with elective resection. Poor-risk patients may be permitted to have 2 or more attacks requiring deflation by rectal tube before resection is advised.

OBSTRUCTION DUE TO ADHESIONS

About 30% of small bowel obstructions are caused by adhesions secondary to previous surgery, peritonitis, or trauma. Strangulation occurs in 10-20% of cases of obstruction which are caused by long-standing adhesions. Freshly formed adhesions, such as those developing in the early postoperative period, are less frequently associated with bowel necrosis. Adhesions rarely produce colonic obstruction.

Treatment of adhesive obstruction is similar to that for mechanical obstruction (see p. 267). In view of the low incidence of strangulation and the frequently satisfactory response, obstruction caused by early postoperative adhesions is often given an intensive trial of intubation before surgery is advised.

OBSTRUCTION DUE TO NEOPLASM

Neoplasms of the Small Intestine.

Neoplasm of the small bowel is a relatively rare cause of obstruction. The lesions which occur include carcinoma, lymphoma, leiomyosarcoma, carcinoid, and benign tumors such as adenoma, leiomyoma, and lipoma. Symptoms are often those of chronic or recurrent obstruction. Intussusception of the lesion may precipitate acute obstruction. When intussusception occurs in an adult, a benign or malignant neoplasm should always be suspected as the exciting cause. Treatment is by resection.

Neoplasms of the Colon and Rectum.

Carcinoma is the most frequent cause of large bowel obstruction (see p. 265). The ultimate treatment is by resection. The immediate problem, however, is decompression of the proximal colon when significant obstruction is present. The place of intubation in the management of partial neoplastic obstruction of the colon has already been mentioned (see p. 268). When obstruction is

complete and unremitting, surgical decompression of the colon is
required prior to resection. Transverse colostomy or cecostomy
is employed for severe obstruction located at or distal to the splenic
flexure. Tumors which completely obstruct the proximal half of
the colon usually require a preliminary cecostomy or side-to-side
ileocolostomy. In selected cases, primary resection of the right
colon is feasible after intestinal intubation in spite of high-grade
obstruction.

Abdominal Carcinomatosis.

Obstruction by peritoneal carcinomatous implants is not un-
common. It can occasionally be palliated temporarily by intubation,
limited resection, or entero-enterostomy around the point of ob-
struction.

MISCELLANEOUS CAUSES OF OBSTRUCTION

Intussusception.

This condition is common in children (see p. 155). In adults it
is rare and is usually caused by a benign or malignant tumor of the
bowel. Incomplete or intermittent obstruction may be present, and
bloody mucus may be found in the rectum. Early operation is re-
quired.

Obturation.

Obturation obstruction results when a foreign body is unable to
pass through the bowel. Gallstones (gallstone ileus), bezoars, in-
gested or inserted foreign bodies, worms, and fecal impaction are
common causes. All kinds of obturation except fecal impaction
most frequently involve the small bowel.

Gallstone ileus is an important syndrome which usually occurs
in elderly, obese women, half of whom have a past history of gall-
bladder disease. A fistula forms between the gallbladder and duo-
denum for passage of the stone. Intestinal gas which enters through
the fistula can almost always be seen in the biliary tree by x-ray if
searched for with care. The obstructing gallstone can also be
found on x-ray in about half of cases. Treatment is by prompt lapa-
rotomy to remove the stone. The cholecystoduodenal fistula should
not be disturbed at the operation for obstruction.

Obstruction by worms (usually Ascaris lumbricoides) occurs
predominantly in children under 10 years of age. The site of ob-
struction is most frequently in the lower ileum. Symptoms may be
acute or chronic. Diagnosis is made by finding ova in the stools
and by thin barium study of the bowel, which may outline the worms.
Distention is controlled by intubation, and the worms are removed
by vermifuge therapy. Operation should be avoided if possible since
worms migrate through the suture lines of enterotomies.

Inflammatory Disorders.

The most common inflammatory lesions which cause obstruc-
tion are regional enteritis (see p. 291), tuberculosis, and amebi-
asis. Treatment must be individualized, and operation for acute
obstruction is avoided by intubation if possible. In the case of
tuberculosis and ameboma, which usually involve the ileocecal

region and for which specific drug therapy is available, the differentiation from carcinoma is important but may be difficult.

MESENTERIC THROMBOSIS AND EMBOLISM

Infarction of the bowel is the result of occlusion of mesenteric veins or major arteries. The relative incidence of the 2 types is probably about the same. Arterial occlusion may be caused by thrombosis of an arteriosclerotic vessel or by embolism. The superior mesenteric artery is usually involved in those cases associated with gangrene. Occlusion of the inferior mesenteric artery (usually asymptomatic) is not uncommon in aneurysm or severe atherosclerosis of the aorta. Mesenteric venous thrombosis may follow appendicitis, strangulated hernia, and abdominal trauma or surgery. It may also occur spontaneously without antecedent cause.

Clinical Findings.

These patients are usually elderly or have cardiac disease. In arterial occlusions, especially embolism, onset is sudden, with severe abdominal pain, vomiting, diarrhea, blood in the stools, and shock. In venous thrombosis the onset is frequently more gradual, with relatively mild symptoms and signs until gangrene is marked. In both types of mesenteric involvement, the abdomen is tender, peristalsis is diminished or absent, and distention develops gradually. Leukocytosis is variable, but tends to be marked; white counts above 30,000 are not uncommon. Temperature is at first normal but rises as gangrene and peritonitis progress. The diagnosis may be suggested on x-ray by the appearance of a single loop of distended, thickened bowel and evidence of peritoneal fluid. Recovery of bloody fluid via abdominal tap (see p. 259) indicates the presence of infarcted bowel.

Treatment.

These patients should be explored with minimal delay, and resection of gangrenous bowel with end-to-end anastomosis should be performed if possible. Unfortunately, the entire small bowel and right colon are frequently infarcted, in which case resection is usually impossible or fatal. When infarction is due to embolism or localized thrombosis of the superior mesenteric artery, embolectomy or thrombectomy may be feasible and should be attempted. Anticoagulants have no place in the preoperative treatment of acute mesenteric occlusion.

Prognosis.

The patient is often moribund when first seen. The mortality for mesenteric thrombosis and embolism is 75-100% in reported series. If it is necessary to resect more than 50% of the small bowel because of gangrene, diarrhea and nutritional deficiency may become a chronic problem.

PARALYTIC ILEUS

In paralytic (adynamic) ileus the obstruction is due to inhibition of intestinal motility; there is no organic obstruction. A mild degree of paralytic ileus occurs after every laparotomy, but recovery normally takes place within 2-3 days unless peritonitis develops. Other lesions commonly associated with paralytic ileus are back injuries, fractured vertebrae or ribs, retroperitoneal and intra-abdominal hemorrhage, renal colic, pulmonary lesions such as pneumonia or pneumothorax, torsion of the ovary, testicle, or omentum, CNS diseases, and severe acute infections. Electrolyte imbalance, particularly hypokalemic alkalosis, will cause or aggravate ileus. Distention of the bowel tends to perpetuate ileus.

Clinical Findings.
There is a history of recent operation, injury, or other precipitating condition. Vomiting usually occurs, and obstipation is the rule. Peristalsis is markedly diminished or absent. The abdomen is distended and may or may not be tender, depending upon underlying factors. Plain films of the abdomen show gaseous distention of both colon and small bowel. This observation is helpful in excluding mechanical block in which distention is proximal to the obstruction. When paralytic ileus persists in spite of treatment, suspect sepsis within the abdomen, electrolyte derangement, or an element of mechanical obstruction.

Treatment.
Most cases of ileus are postoperative, mild, and responsive to restriction of oral intake. As the ileus subsides, the diet is liberalized. Supplementary parenteral fluids should be given until oral intake is adequate.

Severe paralytic ileus requires complete restriction of oral intake, parenteral feeding, and gastrointestinal suction with either a gastric or a long intestinal tube (see p. 268). The latter is more effective if it can be passed into the small bowel. When mechanical obstruction and peritonitis can be ruled out, vasopressin (Pitressin®) may be of value in stimulating peristalsis.

Ileus may rarely be extreme and fail to respond to conservative measures. Marked distention may be a factor in perpetuating the condition. If the patient is deteriorating, abdominal exploration for the following purposes is occasionally necessary: (1) to deflate the gastrointestinal tract by aspiration of the bowel with a catheter passed through a small enterotomy; (2) to maneuver a long intestinal tube through the pylorus into the small bowel; (3) to establish a cecostomy, if necessary; and (4) to rule out mechanical obstruction or intra-abdominal abscess. These procedures serve to relieve distention and prevent its recurrence while the cause of the ileus is being brought under control.

APPENDICITIS

Appendicitis is initiated by obstruction of the appendiceal lumen by a fecalith, inflammation, foreign body, or neoplasm. Obstruction is followed by infection, edema, and frequently infarction of the appendiceal wall. Intraluminal tension develops rapidly and tends to cause early mural necrosis and perforation. All ages are affected, but appendicitis usually occurs in persons between 10 and 30 years of age and is more common in males.

Clinical Findings.

Appendicitis is one of the commonest causes of acute surgical abdomen. The symptoms and signs usually follow a fairly stereotyped pattern, but appendicitis is capable of such protean manifestations that it should be considered in the differential diagnosis of every obscure case of intra-abdominal sepsis.

A. "Classical" Appendicitis:

1. Symptoms - An attack of appendicitis usually begins with epigastric or periumbilical pain associated with 1-2 episodes of vomiting. Within 2-12 hours the pain shifts to the right lower quadrant, where it persists as a steady soreness which is aggravated by walking or coughing. There is anorexia, moderate malaise, and slight fever. Constipation is usual, but diarrhea occurs occasionally.

2. Signs - At onset there are no localized abdominal findings. Within a few hours, however, progressive right lower quadrant tenderness can be demonstrated; careful examination will usually identify a single point of maximum tenderness. The patient can often place his finger precisely on this area, especially if asked to accentuate the soreness by coughing. Light percussion over the right lower quadrant is helpful in localizing tenderness. Rebound tenderness and spasm of the overlying abdominal muscles are usually present. Psoas and obturator signs, when positive, are strongly suggestive of appendicitis. Rectal tenderness is common and, in pelvic appendicitis, may be more definite than abdominal tenderness. Peristalsis is diminished or absent. Slight to moderate fever is present.

3. Laboratory findings - Moderate leukocytosis (10,000-20,000) with an increase in neutrophils is usually present. It is not uncommon to find a few red cells on microscopic examination of the urine, but otherwise the urinalysis is not remarkable.

4. X-ray findings - There are no characteristic changes on plain films of the abdomen.

B. Factors Which Cause Variations From the "Classical" Clinical Picture:

1. Anatomic location of appendix - Abdominal findings are most definite when the appendix is in the iliac fossa or superficially located. When the appendix extends over the pelvic brim, the abdominal signs may be minimal, greatest tenderness being elicited on rectal examination. Right lower quadrant tenderness may be poorly localized and slow to develop in retrocecal or retroileal appendicitis. Inflammation of a

high-lying lateral appendix may produce maximal tenderness in the flank. Bizarre locations of the appendix may rarely occur in association with a mobile or undescended cecum; in such cases symptoms and signs may be localized in the right upper or the left lower quadrant.

2. Age -
   a. Infancy and childhood - In infancy appendicitis is relatively rare, but when it occurs the diagnosis is difficult because of the problem of interpretation of history and physical findings. The disease tends to progress rapidly and, when rupture occurs, to result in generalized peritonitis because of poor localizing mechanisms.
   b. Old age - Elderly patients frequently have few or no prodromal symptoms. Abdominal findings may be unimpressive, with slight tenderness and negligible muscle guarding until perforation occurs. Fever and leukocytosis may also be minimal or absent. When the white count is not elevated, a shift to the left is significant evidence of inflammation.
   c. Obesity - Obesity frequently increases the difficulty of evaluation by delaying the appearance of abdominal signs and by preventing their sharp localization.
   d. Pregnancy - Appendicitis occurs occasionally in pregnant women, and the incidence is the same in the 3 trimesters. The symptoms and signs of appendicitis are essentially unchanged, but the diagnosis is usually more difficult because of confusion with the complications of pregnancy. The site of maximal tenderness is usually near McBurney's point - even in the third trimester, when the cecum is displaced upward. Early operation is essential; the somewhat increased mortality rate of appendicitis during pregnancy can be ascribed to delay in treatment. Fetal mortality is about 2-5%. Progesterone administration to prevent abortion is ineffective and may cause female pseudohermaphroditism. A high oxygen intake during anesthesia is mandatory. A muscle-splitting (McBurney type) incision is less likely to cause difficulty during labor.

Differential Diagnosis.

A. Acute gastroenteritis is the disorder most commonly confused with appendicitis. In rare cases it either precedes or is coincident with appendicitis. Vomiting and diarrhea are more common. Fever and WBC may rise sharply and may be out of proportion to abdominal findings. Localization of pain and tenderness is usually indefinite and shifting. Hyperactive peristalsis is characteristic. Gastroenteritis frequently runs an acute course. A period of observation usually serves to clarify the diagnosis.

B. Mesenteric adenitis may cause signs and symptoms identical with appendicitis. Usually, however, there are some clues to the true diagnosis. Mesenteric adenitis is more likely to occur in children or adolescents; respiratory infection is a common antecedent; localization of right lower quadrant tenderness is less precise and constant; and true muscle guarding is infrequent.

In spite of a strong suspicion of mesenteric adenitis, it is often safer to advise appendectomy than to risk a complication of appendicitis by procrastination.

C. Meckel's diverticulitis may mimic appendicitis. The localization of tenderness may be more medial, but this is not a reliable diagnostic criterion. Because operation is required in both diseases, the differentiation is not critical. When a preoperative diagnosis of appendicitis proves on exploration to be erroneous, it is essential to examine the terminal 5 feet of the ileum for Meckel's diverticulitis and mesenteric adenitis.

D. Regional enteritis (see p. 291), perforated duodenal ulcer (see p. 284), ureteral colic (see p. 384), acute salpingitis (see p. 358), mittelschmerz, ruptured ectopic pregnancy, and twisted ovarian cyst may at times also be confused with appendicitis.

Treatment.
A. Preoperative Care:
1. Observation for diagnosis - Within the first 8-12 hours after onset the symptoms and signs of appendicitis are frequently indefinite. Under these circumstances a period of close observation is essential. The patient is placed at bed rest and given nothing by mouth. Laxatives are never prescribed. Parenteral fluid therapy is begun as indicated. Narcotic medications are avoided if possible, but sedation with barbiturates or tranquilizing agents is not contraindicated. Abdominal and rectal examinations, WBC, and differential are repeated periodically. Abdominal films (see p. 259) and an upright chest film are obtained on all difficult diagnostic problems. In most cases of appendicitis, the diagnosis is clarified by localization of signs to the right lower quadrant within 24 hours after onset of symptoms.
2. Intubation - Preoperatively, a gastric tube is usually passed. The stomach is aspirated and lavaged if necessary, and the patient is sent to the operating room with the tube in place.
3. Antibiotics - In the presence of marked systemic reaction with severe toxicity and high fever, preoperative administration of antibiotics (e.g., penicillin and streptomycin) is advisable.
B. Surgical Treatment: In uncomplicated appendicitis, appendectomy is performed as soon as fluid imbalance and other significant systemic disturbances are controlled. Little preparation is usually required. Early, properly conducted surgery has a mortality of a fraction of 1%. Morbidity and mortality in this disease stem primarily from the complications of gangrene and perforation which occur when operation is delayed.
C. Postoperative Care: In uncomplicated appendicitis, postoperative gastric suction is usually not necessary. Ambulation is, begun on the first postoperative day. The diet is advanced from clear liquids to soft solids during the second to fifth postoperative days, depending upon the rapidity with which peristalsis and gastrointestinal function return. Parenteral fluid supplements are administered as required. Enemas, except of the small oil retention type, are contraindicated. Mineral oil or other nonirritating laxatives may be given orally at bedtime daily from about the fourth day onward if necessary. Antibiotic

therapy (e. g., penicillin with streptomycin and/or tetracycline) is advisable for 5-7 days or longer if abdominal fluid at operation was purulent or malodorous, if culture was positive, or if the appendix was gangrenous. Primary wound healing is the rule, and the period of hospitalization is usually 1 week. Normal activity can be resumed in 2-3 weeks after surgery in uncomplicated cases, especially if a McBurney type incision was used.

D. **Nonsurgical Treatment:** When surgical facilities are not available, treat as for acute peritonitis (see p. 260). On such a regimen acute appendicitis will frequently subside and complications will be minimized.

Complications.

A. **Perforation:** Appendicitis may subside spontaneously, but it is an unpredictable disease with a marked tendency to progression and perforation. Because perforation rarely occurs within the first 8 hours, diagnostic observation during this period is relatively safe. Signs of perforation include increasing severity of pain and tenderness, and spasm in the right lower quadrant followed by evidence of generalized peritonitis or of a localized abscess. Ileus, fever, malaise, and leukocytosis become more marked. If perforation with abscess formation or generalized peritonitis has already occurred when the patient is first seen, the diagnosis may be quite obscure.

   Treatment of perforated appendicitis is appendectomy unless a well-localized right lower quadrant or pelvic abscess has already walled off the appendix. Supportive measures are as for acute peritonitis (see p. 260).

B. **Generalized Peritonitis:** This is a common sequel to perforation. Clinical findings and treatment are discussed on p. 260.

C. **Appendiceal Abscess:** This is a common complication of untreated appendicitis. Malaise, toxicity, fever, and leukocytosis vary from minimal to marked. Examination discloses a tender mass in the right lower quadrant or pelvis. Pelvic abscesses tend to bulge into the rectum or vagina.

   Abscesses usually become noticeable 2-6 days after onset, but antibiotic therapy may delay their appearance. Appendiceal abscess is occasionally the first and only sign of appendicitis and may be confused with neoplasm of the cecum, particularly in the older age group in whom systemic reaction to the infection may be minimal or absent.

   Treatment of early abscess is by intensive combined antibiotic therapy (e. g., penicillin and streptomycin and/or tetracycline). On this regimen, the abscess will frequently resolve. Appendectomy should be performed 6-12 weeks later. A well-established, progressive abscess in the right lower quadrant should be drained without delay. Pelvic abscess requires drainage when it bulges into the rectum or vagina and has become fluctuant.

D. **Pylephlebitis:** Suppurative thrombophlebitis of the portal system with liver abscesses is a rare complication. It should be suspected when septic fever, chills, hepatomegaly, and jaundice develop after appendiceal perforation. Intensive combined antibiotic therapy is indicated.

E. Other complications include subphrenic abscess (see p. 57) and other foci of intra-abdominal sepsis. Mechanical intestinal obstruction (see p. 265) may be caused by adhesions.

MASSIVE UPPER GASTROINTESTINAL HEMORRHAGE

Massive gastrointestinal hemorrhage is a common emergency. The immediate objectives of management are (1) to control shock and (2) to establish a diagnosis on which definitive treatment can be based.

Etiology.

About 75% of cases are due to peptic ulceration of the duodenum or stomach. Esophageal varices and gastritis are each responsible for about 10% of cases. Gastric neoplasm, hiatus hernia, esophagitis, and miscellaneous disorders account for about 5%.

Clinical Findings.

There is usually a history of sudden weakness or fainting associated with or followed by tarry stools and/or vomiting of blood. Melena occurs in all patients, and hematemesis in over 50%. Hematemesis is especially common in esophageal varices (90%), gastritis, and gastric ulcer. The patient may or may not be in shock when first seen, but he will at least be pale and weak if major blood loss has occurred.

There is usually no pain, and abdominal findings are not remarkable except when hepatomegaly, splenomegaly, or a mass (neoplasm) is present. There may be a past history of peptic ulcer, cirrhosis, or other predisposing disease, but history often gives no clue to the source of bleeding. About half of all patients will have had at least 1 previous hemorrhage.

The etiology of bleeding should be established promptly, if possible, since the decision whether to operate or to continue with medical measures often depends upon the diagnosis. The most critical differentiation is between peptic ulcer and esophageal varices, since emergency surgery is frequently indicated and successful in ulcer but less often indicated or successful in varices. Specific diagnosis is of value also because of the difficulties of entering the abdomen in search of an unknown bleeding point.

The principal diagnostic procedures in the investigation of upper gastrointestinal bleeding are as follows:

A. Laboratory Findings: When cirrhosis with bleeding varices cannot be ruled out, the following blood studies should be obtained immediately.

1. Sulfobromophthalein sodium (BSP) retention test - Normal BSP retention practically excludes cirrhosis severe enough to cause varices. However, because retention (15% or greater) occurs frequently in massive hemorrhage in the absence of cirrhosis, a positive test is not conclusive evidence of liver disease.

      2. Blood ammonia level - Blood ammonia is almost always
elevated in cirrhotics with bleeding esophageal varices
within 1-2 hours after bleeding begins.

      3. Prothrombin time - This test should be performed in all
cases of suspected liver disease.

B. X-ray Findings: The cause of upper gastrointestinal bleeding
can be demonstrated on x-ray in about 75% of cases. When the
diagnosis is in doubt, emergency barium examination of the
upper gastrointestinal tract should be done immediately. The
examination is only postponed if there is active bleeding or
shock.

C. Esophagoscopy: When varices are suspected in spite of nega-
tive x-rays, esophagoscopy is useful. The procedure can be
done on the operating table just prior to laparotomy if necessary.
When both varices and peptic ulcer are seen on x-ray, esoph-
agoscopy may help to decide which is bleeding.

Treatment.

A. General Measures: The patient should be under the observation
of both an internist and a surgeon from the outset. Bed rest,
mild sedation if necessary, and regular recording of BP,
pulse, respiration, temperature, and urine output are insti-
tuted. Vitamin K_1 is given empirically. Treatment of shock
by blood transfusion is begun without delay (see p. 9). Hct.
or Hgb. determinations are done every few hours until stabi-
lized. The objective of blood replacement is to relieve shock
and restore the Hct. to 35% and the Hgb. to 12 Gm. /100 ml.

B. Medical Measures: Acid peptic digestion is a causative or ag-
gravating factor in most cases of massive upper gastrointes-
tinal hemorrhage, including varices. Bland feedings and oral
medications for ulcer are begun as soon as shock and nausea
have subsided. Continued slight bleeding is no contraindication
to the following regimen:

      1. Diet - Hourly feedings (on the hour) around the clock of 90
ml. of half milk and half cream (Sippy Stage I). Three to
6 Gm. of sodium chloride may be added to each quart of
milk and cream mixture to prevent salt depletion. The diet
may be advanced over the next few days as tolerated to
puréed bland foods (see p. 94).

      2. Antacids - Aluminum hydroxide gel—magnesium trisilicate
mixture (Maalox®, Gelusil®, etc.), 15-30 ml., is given
hourly (on the half-hour), alternating with the milk and
cream mixture.

      3. Other medications indicated include anticholinergics (see
p. 612) and mild barbiturate sedation (see p. 607).

C. Management of Bleeding Esophageal Varices: When varices
are the cause of bleeding, special measures are indicated (see
p. 321).

D. Indications for Emergency Operation: Except when esophageal
varices are the cause of bleeding, emergency surgery to stop
active bleeding should be considered under any of the following
circumstances:

1. When the patient has received 1000 ml. or more of blood but shock is not controlled or recurs promptly.
2. When acceptable BP and Hct. cannot be maintained with a maximum of 500 ml. of blood every 8 hours.
3. When bleeding is slow but persists more than 2-3 days.
4. When bleeding stops initially but recurs massively while the patient is receiving adequate medical treatment.
5. When the patient is over 50. It has been shown that the death rate from exsanguination in spite of conservative measures is greater in the older age group and rare in patients under 40. Massive bleeding is less well tolerated and is less likely to stop in older patients, who will therefore require operative intervention more frequently.

E. Intragastric Cooling: Local gastric hypothermia by means of circulation of cold fluid through an intragastric balloon has proved very effective in controlling massive hemorrhage and is worthy of trial in selected cases (Wangensteen & others, Surgery 44:265, 1958).

Prognosis.

The over-all mortality of about 14% indicates the seriousness of massive upper gastrointestinal hemorrhage. Fatality rates vary greatly, depending upon the etiology of the bleeding. Hemorrhage from duodenal ulcer causes death in about 3% of treated cases, whereas in bleeding varices the mortality rate may be as high as 50%.

DISEASES OF THE ESOPHAGUS

Dysphagia is the most frequent complaint in esophageal disease. This symptom, no matter how mild, should always be investigated by esophageal x-rays with contrast medium. If there is a lesion which requires further evaluation or if roentgenography is negative and dysphagia continues, esophagoscopy is advisable. Biopsy specimens can be obtained through the esophagoscope when indicated.

ESOPHAGEAL TRAUMA

Esophageal injury caused by an external crushing force is associated with serious damage to other viscera; penetration of the esophagus by missiles, sharp objects, or instruments may cause little injury to other structures. Rupture of the lower esophagus sometimes occurs as a result of violent vomiting or perforation of an esophageal ulcer. Immediate diagnosis and surgical repair are essential to prevent overwhelming mediastinal and pleural infection, although small rents produced by instrumentation (e.g., esophagoscopy, bougienage) may be successfully treated by intensive penicillin and streptomycin therapy. A swallow of soluble contrast medium, e.g., Methylglucamine Diatrizoate, N.N.D. (Gastrografin®) (never barium), under fluoroscopy and esophagoscopy, are useful diagnostic procedures in doubtful cases.

See p. 147 for chemical burns of esophagus.

DIVERTICULA OF THE ESOPHAGUS

Pulsion diverticula of the esophagus due to herniation of mucosa and submucosa through a weakened portion of the muscle wall may occur either at the pharyngoesophageal junction or just above the diaphragm. Traction diverticula, resulting from the traction of an adjacent inflammatory process (usually tuberculous adenitis), occur occasionally at the midthoracic level but rarely cause symptoms or require treatment.

Pharyngoesophageal (Hypopharyngeal) Diverticula.

These project from the posterior wall of the hypopharynx just above the cricopharyngeus muscle and usually veer to the left of the midline. They rarely occur before middle age. Symptoms and signs consist of dysphagia, regurgitation of food (often that eaten several days previously), and gurgling noises in the neck on swallowing or on pressure over the diverticulum. Cough from tracheal irritation is common; aspiration pneumonia may result from overflow of the diverticulum; and nutritional deficiency may develop if there is serious interference with swallowing. Treatment is by one-stage excision.

Supradiaphragmatic Diverticula.

These are rare. Dysphagia, regurgitation of previously ingested food, and substernal or back pain are the chief complaints. Diagnosis is made readily on barium study. Treatment is by one-stage transthoracic excision.

CARDIOSPASM

In idiopathic dilatation of the esophagus (achalasia, cardiospasm, mega-esophagus, etc.) the entire esophagus down to its distal few cm. may assume huge proportions. The terminal, relatively narrowed portion may show either atrophy or hypertrophy of the muscle layers, but there is no actual obstruction and the defect appears to be a dystonia of the esophagus related to absence or diminution of the ganglion cells of Auerbach's plexus. All ages are affected, but the condition is most common in early adult life.

Clinical Findings.

Dysphagia, regurgitation of previously ingested food, and epigastric or subxiphoid pain are common symptoms. Overflow from the esophagus may produce aspiration pneumonia. Some patients become severely malnourished. Barium study of the esophagus is usually diagnostic, but carcinoma of the cardiac end of the stomach occasionally causes esophageal obstruction with proximal dilatation. When the diagnosis is in doubt, esophagoscopy and biopsy of the cardia are mandatory.

Treatment.

Low-residue diet (see p. 97) and anticholinergic drugs (see p. 612) may suffice to control mild symptoms. About 80% of patients can be permanently relieved by 1 or more dilatations with the hydrostatic dilator, but this method of treatment requires skill

and experience. Approximately 20% of cases require operation. The Heller procedure (myotomy of the distal, narrow portion of the esophagus) is the least dangerous and most successful. Other operations which result in regurgitation of gastric contents into the lower esophagus are complicated by a high incidence of peptic esophagitis, which may be more disabling than the original disease.

PEPTIC ESOPHAGITIS

Inflammation of the distal esophagus caused by reflux of gastric juice produces heartburn and substernal pain on swallowing. Ulceration, stricture, hemorrhage, and, rarely, perforation are the major complications. There are 2 predisposing conditions: the peptic ulcer diathesis and hiatus hernia. Diagnosis is made by x-ray and esophagoscopy.

When peptic disease is an aggravating factor, dietary and drug therapy should be given as for peptic ulcer (see p. 284) and the patient should sleep on pillows in the semi-reclining position. Small hiatus hernia with mild esophagitis is treated similarly; larger and more symptomatic hernias require surgical repair. Peptic esophagitis associated with shortening of the esophagus, cicatricial stricture, or traction-type hiatus hernia is best managed by an ulcer regimen and dilatation with mercury bougies. Intractable cases may respond to partial distal gastric resection to reduce gastric acidity (Wangensteen), esophagogastrectomy (Sweet), resection of the stricture with esophagojejunostomy (Allison), or hiatus hernia repair, 50% gastrectomy, and vagotomy (Bartlett).

ESOPHAGEAL NEOPLASMS

Dysphagia is the universal early symptom of neoplastic diseases of the esophagus. X-ray and esophagoscopy with biopsy establish the diagnosis.

Benign Tumors.
Benign esophageal tumors are rare; leiomyomas (most common), fibromas, lipomas, and cysts are treated by local excision.

Malignant Tumors.
Epidermoid carcinoma is the commonest esophageal tumor. Lesions above the lower third spread to regional nodes (beyond the limits of surgical removal) so early that resection is rarely more than palliative. Surgical resection may occasionally be successful but, in general, x-ray therapy is the preferred method of management in tumors above the lower third. Lesions of the lower third of the esophagus should be removed surgically when there is no evidence of metastasis. The five-year survival rate after resection of carcinoma of the lower third of the esophagus is about 30%. Obstruction of the esophagus by incurable carcinoma can often be relieved temporarily by x-ray therapy with or without esophageal dilatations. It may be possible for feeding purposes to pass a small tube through the lesion into the stomach by endoscopy when severe

dysphagia prevents adequate oral intake. Gastrostomy may be justified when other methods of feeding fail and the general condition of the patient is good.

Adenocarcinomas of the cardiac end of the stomach frequently invade the distal esophagus and may be confused with esophageal carcinoma.

DISEASES OF THE STOMACH AND DUODENUM

PEPTIC ULCER

Ten per cent of people have peptic ulcer during their lifetime. It is most common in young and middle aged adults, and men are affected 4 times as often as women. Diagnoses based on x-ray examination reveal a 9:1 preponderance of duodenal over gastric ulcer, but autopsy diagnoses show an equal incidence of the 2 types.

The etiology is not known, but the action of acid and pepsin on the gastroduodenal mucosa of constitutionally susceptible persons undoubtedly plays a role. Acute ulcers may occur following prolonged administration of corticotropin or corticosteroids, reserpine, phenylbutazone, and salicylates; intracranial injury or disease, massive infection, severe burns, and other forms of stress.

Peptic ulcer must be suspected in any case of chronic dyspepsia. The most characteristic complaint is gnawing, "hunger-like" epigastric pain occurring one-half to several hours after eating which is relieved by food or alkalies. Emotional stress is an aggravating factor. Remissions and relapses are common. The physical examination is usually negative, although mild tenderness in the epigastrium may be present. Diagnosis is established by gastrointestinal x-rays.

Medical Management.

The conservative management of peptic ulcer consists of bland diet, antacids, anticholinergics, mild sedatives, rest, and psychotherapy.

A. Diets: These range from the strict Sippy Stage I regimen for acute and complicated ulcers to the more liberal six-meal bland diet (see p. 95).

B. Antacids: (See p. 612.) Aluminum hydroxide–magnesium trisilicate combinations are most effective (e.g., Maalox®, Gelusil®). During the first stage of management of severe ulcers, the antacid, 15-30 ml., is given hourly on the half hour and the feeding (90 ml.) of milk and cream is given hourly on the hour throughout the day and when awake at night. When used with an ambulatory regimen and bland diet, the antacid is most conveniently given in tablet form 90 minutes after meals and at any other time that ulcer distress occurs.

C. Anticholinergic Agents: (See p. 612.) Tincture of belladonna is preferred for oral use; atropine when parenteral administration is desirable.

D. **Other Measures:** Regular eating habits and adequate rest are essential. Bed rest and hospitalization may be advisable in severe cases. Mild barbiturate sedation (see p. 607) is often helpful. Coffee and other caffeine-containing beverages, alcohol, and tobacco are eliminated. Mental and emotional conflicts should be treated by psychotherapy.

Indications for Surgical Treatment.

Ninety per cent of peptic ulcers respond well to medical management; the remainder require surgical treatment. Indications for surgery are as follows: (1) intractability, (2) perforation, (3) hemorrhage, (4) obstruction, and (5) gastric ulcer with suspicion of malignancy.

A. **Intractability:** When symptoms persist or recur frequently in spite of appropriate medical therapy, or when the patient is unable for some reason to cooperate in an effective program, surgical treatment should be considered.

B. **Perforation:** This complication occurs in about 2% of ulcer patients. There is usually (but not always) a past history of ulcer symptoms. Prompt treatment is essential; the over-all mortality for perforated ulcer ranges from 5-10%, but is only 1-2% with early treatment.

 1. **Diagnosis** - The onset of severe epigastric or generalized abdominal pain is sudden. The pain may be referred to the top of 1 or both shoulders (phrenic irritation). Boardlike rigidity of the abdomen with epigastric or generalized tenderness and absent or markedly diminished peristalsis are typical. Occasionally, localization of maximum tenderness in the right lower quadrant suggests appendicitis (see p. 275). The patient tends to lie still with knees flexed to minimize the pain. Temperature and BP may be subnormal immediately following perforation, but fever develops gradually. Leukocytosis is moderate to marked, and serum amylase and urinary diastase may be moderately elevated. Pneumoperitoneum is noted on upright chest or lateral decubitus abdominal films in about two-thirds of patients. Acute pancreatitis, acute cholecystitis, appendicitis, perforation of another abdominal viscus, diaphragmatic pleurisy, and coronary thrombosis are the principal disorders which must be considered in the differential diagnosis.

 2. **Emergency measures** - A gastric tube is passed immediately and placed on constant suction; treat as for acute peritonitis (see p. 260). Operation, if indicated, should be carried out as soon as the patient's condition has been stabilized by fluid replacement and other measures.

 3. **Treatment** - There are 3 possible types of therapy: (1) nonoperative treatment, (2) simple closure, and (3) emergency gastric resection. Most patients are best managed by simple closure of the perforation.

  a. **Nonoperative treatment** - Nonoperative treatment consists of constant gastric suction, antibiotics, and other measures for acute peritonitis (see p. 260). Other than those patients who are too ill for any type of surgical procedure or whose ulcers perforate in circumstances where surgical facilities are not available, only a very small group

are best treated by nonoperative methods: (1) patients seen 24 hours or more after perforation in whom abdominal findings suggest that the perforation has already sealed; and (2) patients who show no free air on x-ray and in whom the differential diagnosis cannot be made between perforated ulcer and a disorder such as myocardial infarction in which surgery is contraindicated.

b. Simple closure of the perforation - This is the safest and most widely applicable mode of treatment and should be employed except when nonoperative treatment or gastrectomy is preferable.

c. Emergency gastrectomy - If the patient is a good surgical risk, the perforation is recent (less than 12 hours), and the operative findings indicate that contamination is minimal and resection will not be difficult, immediate gastrectomy should be performed in the following cases:

(1) Perforated gastric ulcer (8-10% are malignant).

(2) Perforation with massive hemorrhage or organic obstruction.

(3) Severe ulcer diathesis, as evidenced by previous hemorrhage or perforation, chronic ulcer symptoms despite medical therapy, or perforation during an intensive medical regimen.

4. Postoperative and after-care - Postoperatively the patient is treated for acute peritonitis (see p. 260). Diet is advanced as tolerated to a six-meal bland diet with antacids and anticholinergics. Intensive medical management for ulcer should be carried out for at least 3 months and as long thereafter as the symptoms warrant.

5. Prognosis - About one-third of patients treated by simple closure or nonoperative methods will be asymptomatic or will have only mild symptoms in the future; one-third will have symptoms requiring medical management; and one-third will ultimately require surgery.

C. Massive Hemorrhage From Peptic Ulcer: See p. 279.

D. Pyloric Obstruction:

1. Diagnosis - There is usually a past history of ulcer symptoms culminating in recurrent vomiting, frequently of food eaten at the previous meal. Weight loss, dehydration, metabolic alkalosis, and hypokalemia are late signs of depletion. Tympanitic epigastric fullness may be present, with visible peristalsis in the distended stomach. The diagnosis is confirmed by barium meal: pyloric obstruction is present if there is significant gastric retention of barium in 6 hours or any residual barium after 24 hours. Obstructing neoplasm must be excluded. A fasting gastric content of more than 100 ml. on aspiration is usually indicative of obstruction.

Pyloric obstruction due to edema and spasm will respond to medical measures. It must be distinguished from obstruction due to scarring, which requires surgical treatment.

2. Treatment -

a. Medical measures - These should be given a trial in most cases, with the patient at bed rest in a hospital if possible.

     (1) Diet - Begin with Sippy Stage I diet and advance to
        Sippy II, III, and IV as tolerated.
     (2) Medications - Give sedative, antacid, and anticholinergic drugs as for uncomplicated ulcer.
     (3) Fluids and feeding - Oral intake will at first usually
        require intravenous supplementation. Electrolyte
        balance should be reestablished (see p. 99).
     (4) Gastric aspiration and indications for operation - With
        the patient on Sippy I diet, aspirate the stomach in the
        morning and again in the evening and measure intake
        and output carefully. If the patient is improving, intake will exceed output by a little more each day. When
        less than 100 ml. are recovered by aspiration, aspirations are made only p.r.n. and the diet gradually
        advanced. If the volume of aspirated residuum fails
        to decrease rapidly over a 3-5 day period, cicatricial
        stenosis is present and surgery is indicated.

  b. Surgical treatment - The stomach should be thoroughly
     decompressed by constant gastric tube drainage or suction for a few days preoperatively. Gastric resection or
     other definitive operation for ulcer should be done. Gastroenterostomy is a compromise procedure advisable
     only in elderly patients with long-standing cicatricial obstruction and low free acid content who will not tolerate
     more extensive surgery. Such cases are rare.

E. Gastric Ulcer: The symptoms of duodenal and gastric ulcer
are similar. However, gastric ulcer tends to occur in older
persons and is less related to hyperacidity than duodenal ulcer.
Normal or low free acid is common in gastric ulcer. Eight to
20% of benign-appearing gastric ulcers prove to be carcinoma.
For this reason the patient with gastric ulcer should be treated
intensively and kept under close observation. Medical management is the same for gastric as for duodenal ulcer.

1. Signs of malignancy in gastric ulcer -
  a. Achlorhydria - Absence of free acid after histamine is
     virtually never found in benign ulcer.
  b. Malignant cytology - The finding of malignant cells by
     Papanicolaou smear of gastric washings indicates the
     presence of neoplasm.
  c. X-ray appearance - A malignant ulcer frequently appears
     as a filling defect in a crater or a mass. Location on the
     greater curvature is highly suggestive of cancer. The
     wall neighboring a malignancy often has diminished pliability. The size of the ulcer is of little diagnostic significance, as benign lesions can be very large.
  d. Gastroscopy - Benign ulcers are cleanly punched-out
     with a smooth base; malignant ulcer may have a heaped-up, irregular margin, and there may be bleeding from
     the base.
  e. Failure of response to treatment - Many malignant gastric ulcers satisfy none of the above criteria. Failure to
     respond rapidly to 3 weeks of intensive medical therapy
     for ulcer is strong evidence for carcinoma. On the other
     hand, good response does not rule out malignancy since
     ulcer in cancer can heal over temporarily. For this

reason, all gastric ulcers should have follow-up x-ray examinations about 3 weeks, 6 weeks, 3 months, and 6 months from the beginning of treatment and thereafter as symptoms indicate. Recurrence of ulcer under medical management suggests carcinoma; about 16% of recurrent gastric ulcers prove to be neoplastic.

2. **Indications for operation** - Gastric resection is indicated when gastric ulcer exhibits any of the above signs of malignancy or fails either to heal completely or to show marked regression after 3 weeks of intensive medical care. In addition, perforation, obstruction, and massive hemorrhage should be treated surgically according to the criteria outlined on pp. 285-287.

Operations for Peptic Ulcer.

The objective of surgical treatment is the marked reduction or elimination of free gastric hydrochloric acid with maximum preservation of gastrointestinal function. The 2 procedures which most successfully meet these specifications are (1) two-thirds to three-quarters distal gastric resection with gastrojejunostomy (Billroth II), and (2) total vagotomy combined with distal hemigastrectomy and gastrojejunostomy. The latter operation is preferred when the patient is poorly nourished and does not have a robust appetite. Two-thirds distal gastrectomy and gastroduodenostomy (Billroth I) may be employed in gastric ulcer. The mortality for these operations when done electively should be no more than 1-2%.

Postoperative Care.

Nothing is given by mouth for 24-48 hours. Diet is then increased gradually, beginning with 30 ml. of fluid per hour (see p. 95). Gastric suction is maintained for 24-48 hours postoperatively, and ambulation is begun on the first postoperative day. Milk of magnesia and mineral oil by mouth (see p. 610) or a low saline enema on the third or fourth postoperative day will aid in establishing bowel regularity if needed.

Early Postoperative Complications.

A. **Duodenal Stump Leakage:** Sudden, severe upper abdominal pain on the third to seventh postoperative day (occasionally later) is characteristic. The patient is prostrated and acutely ill, with rising fever and WBC. Marked rigidity of the upper or entire abdomen is usual, and may be difficult to evaluate because of the presence of the incision. Radiation of pain to the shoulder (phrenic referral) is suggestive of stump or suture-line leakage.

Treatment is by immediate placement of drains or suction in the region of the leak. Attempts to resuture the duodenal stump are unsuccessful. If adequate external drainage is provided and the afferent jejunal loop is unobstructed, the duodenal leak tends gradually to close.

B. **Gastric Retention:** Failure of the gastrojejunostomy or gastroduodenostomy to function promptly occurs to a significant degree in about 5% of cases.

Gastric distention must be avoided during the early postoperative period. The patient's stomach should be aspirated if he feels "full" or nauseous after removal of the tube. Treatment

of gastric retention consists of resuming gastric suction for 24-48 hours. The gastric tube is then left on gravity drainage and the first-day gastrectomy diet is resumed. The tube is clamped for the first half hour after feeding and then unclamped for the remaining half hour. Drainage volume is recorded. The feeding volume is increased and the tube is clamped for longer intervals or removed as volume of drainage diminishes.

Hypernatremia, hypokalemia, and hypoproteinemia are precipitating or aggravating conditions which should be relieved. The functioning of the stoma should be checked by barium x-ray study if obstruction persists more than a few days. Reoperation must be seriously considered if total gastric retention persists more than 10 days, although conservative treatment may be successful after periods of several weeks.

Sequelae of Gastric Surgery.

A. Marginal Ulcers: Marginal ulcers develop in about 5% of cases and may be controlled by a medical ulcer regimen. If this fails, a vagotomy may be done or a more adequate gastrectomy performed. Gastrcjejunocolic fistula is the most serious complication of marginal ulcer and should be suspected when persistent pain or ulcer symptoms, vomiting, diarrhea, and nutritional depletion develop following gastrectomy with gastrojejunostomy. Diagnosis is readily made by gastrointestinal x-rays. Treatment is by resection of the fistula after careful preparation of the patient.

B. Dumping Syndrome: The so-called "dumping syndrome" occurs after 5-10% of resections. It is characterized by the development, shortly after eating, of sweating, warmth, flushing, nausea, palpitation, and faintness. Diarrhea may or may not be present. The etiology is obscure, but the syndrome may be caused by acute reduction in plasma volume or by excitation of autonomic reflexes due to rapid filling of the jejunum. Symptoms can usually be controlled by a regimen consisting of frequent low-residue feedings, avoidance of high-carbohydrate foods, restriction of fluids during meals, and lying down after eating. Anticholinergics (see p. 612), mild sedation (see p. 607), and reassurance may have value.

C. Other Sequelae: Failure to gain or actual loss of weight occurs in 15-30% of patients after an adequate gastric resection. Failure normally to assimilate iron, fat, carbohydrate, or protein may occur. The more radical the gastrectomy, the higher the incidence of these disturbances. Management is with frequent feedings and supplementary iron and vitamin therapy. Diarrhea occurs in about 7% of vagotomized patients and is treated by nonspecific measures (see p. 611).

NEOPLASMS OF THE STOMACH

Benign Neoplasms of the Stomach.

These are rare and usually asymptomatic, although leiomyoma, the commonest benign tumor, may cause obstruction or bleeding. Single or multiple adenomatous polyps are probably precancerous lesions. Lipomas, fibromas, hemangiomas, aberrant pancreatic

tissue, and a variety of other benign lesions have been reported.

All benign gastric neoplasms should be excised so that differentiation from malignancy can be made by microscopic study.

Malignant Neoplasms of the Stomach.

A. Adenocarcinoma: This common lesion is among the most frequent causes of cancer deaths in men. Ninety-five per cent of cases occur in persons past 45 years of age, and two-thirds of the patients are men. Gastric polyps, chronic gastritis, pernicious anemia, and achlorhydria may be precursors. There may be a family history of gastric cancer.

The earliest symptoms are deceptively vague, consisting usually of anorexia or slight dyspepsia. When such complaints (or other forms of indigestion) last more than 3 weeks in a previously well person over 45 years of age, carcinoma of the stomach should be suspected and gastrointestinal x-rays obtained. The physical examination is negative except in advanced disease, when weight loss, anemia, an abdominal mass, and progressive gastric malfunction commonly develop.

Surgical resection is the only curative treatment. Signs of metastatic disease include a hard, nodular liver, enlarged left supraclavicular (Virchow's) nodes, skin nodules, ascites, rectal shelf, and x-ray evidence of osseous or pulmonary metastasis. If none of these are present and there is no other contraindication to operation, exploration is indicated. The presence of an abdominal mass is not a contraindication to laparotomy, since bulky lesions can often be totally excised. The operative mortality in partial gastrectomy for cancer is about 6%, with a 27% five-year survival rate; for total gastrectomy, the mortality is about 9% with a 14% five-year survival rate. Palliative resection or gastroenterostomy is occasionally helpful in pyloric obstruction. X-ray therapy is of no value.

B. Leiomyosarcoma: This is a rare tumor with a tendency to hemorrhage. It usually produces little gastric distress, even though large, and anemia may be the chief sign. Leiomyosarcomas grow slowly, are slow to metastasize, and seldom recur after adequate excision; the prognosis is therefore much more favorable than with carcinoma.

C. Lymphosarcoma: The signs and symptoms of lymphosarcoma are similar to those of carcinoma. The differential diagnosis is usually made only after microscopic examination. Resection should be followed by radiation therapy. The resectability and survival rates for lymphosarcoma are higher than for carcinoma.

DUODENAL DIVERTICULUM

True duodenal diverticula are noted frequently as an incidental finding on x-ray examination, usually on the mesenteric border. They rarely cause symptoms, but may do so if retention within the sac results in infection, ulceration, hemorrhage, or perforation. Duodenal diverticulum usually requires no treatment.

DISEASES OF THE JEJUNUM AND ILEUM

EXTERNAL SMALL BOWEL FISTULAS

Clinical Findings.

The commonest causes of external small bowel fistula are accidental or surgical trauma, leaking intestinal suture lines or duodenal stump closure, strangulation or obstruction of the small intestine, peritoneal sepsis, and regional enteritis. The fistula can be demonstrated by the appearance in the wound of methylene blue or indigo carmine administered by mouth; or by x-ray examination after ingestion of barium or after injection of methylglucamine diatrizoate (Gastrografin®) or iodized oil (Lipiodol®) into the fistulous tract.

Treatment.

Conservative measures are employed to support the patient during spontaneous closure or, in large or distally obstructed fistulas, in preparation for laparotomy. If nutrition and fluid balance are well maintained, gradual closure over a period of weeks can be expected in most cases.

A. Maintenance of Fluid and Nutritional Balance: High jejunal fistulas may seriously deplete the patient within a few hours. Strict accounting of fluid intake and output and quantitative replacement are essential (see p. 99).

B. Local Care of the Fistula: Collection of fistulous drainage and prevention of skin excoriation by intestinal secretions are major problems. A suction catheter over or in the fistula can often be employed. Occasionally it is possible to glue a plastic ileostomy bag to the skin to pick up drainage. It may be advantageous to place the patient prone on a Bradford frame and allow the fluid to drop down into a basin when skin irritation is otherwise uncontrollable. From the outset the skin around a fistula should be carefully protected by application of silicone ointment (e.g., Silicote®), zinc oxide ointment, 10% powdered aluminum in zinc oxide ointment, or a watery paste of kaolin or Fuller's earth. Exposure of the skin to the drying effect of a desk lamp several times daily is useful.

C. Surgical Treatment: Continuous copious drainage caused by a large fistula or by obstruction of the bowel distally is an indication for surgical repair. Resection of the fistula or enteroenterostomy around it, with later resection of the involved bowel, is usually required. Preoperative placement of a Miller-Abbott or other long intestinal tube through the nose down to the fistula is an aid in identifying the proximal limb of the fistula at operation.

REGIONAL ENTERITIS

This granulomatous inflammatory disease of unknown etiology may occur in any portion of the gastrointestinal tract, but is most commonly seen in the terminal ileum. Multiple, segmental involvement is not unusual. The highest incidence is between the ages of

20 and 40. Marked thickening of the bowel wall and adjacent mesentery are characteristic pathologic features. Recurrence, fistula formation (internal or external), local perforation with abscess, and malabsorption are the most frequent complications. Clinically, regional enteritis is usually a chronic disease, but it may also occur in an acute form.

Clinical Findings.

The acute form often presents as an acute surgical abdomen with fever, leukocytosis, and abdominal tenderness (usually in the right lower quadrant). There may be a past history of diarrhea and intestinal colic which is helpful in the differentiation from appendicitis. An indefinite boggy mass may be palpable. If regional enteritis is suspected, an emergency barium enema with reflux into the ileum should be obtained and may establish the diagnosis.

Patients with chronic regional enteritis usually give a history of recurrent, generalized abdominal pain, diarrhea, and weight loss. Fever and anemia are also common. In many cases an appendectomy will have been done because of abdominal pain. Occasionally there is a palpable mass or rectal bleeding. Segmental narrowing of the bowel lumen is often demonstrable on gastrointestinal x-rays, and is characteristic when present.

Parasitic infestation, tuberculous enteritis, and neoplasm must be considered in the differential diagnosis.

Treatment.

A. Medical Measures: The objective of conservative management is to maintain nutrition by a high-caloric, low-residue, bland diet with vitamin and iron supplements. Diarrhea is controlled with nonspecific medications (see p. 611). Antibiotic therapy is useful only for perforation and abscess formation. Corticosteroids (see p. 607) are reserved for use in preoperative preparation of the severely ill patient.

B. Surgical Treatment: A mistaken diagnosis of appendicitis may lead to laparotomy in the acute stage. The appendix is removed unless regional enteritis involves the cecum or neighboring ileum, in which case appendectomy is not done for fear of fistula formation. Resection of the acute process in the bowel is usually not indicated. Permanent remissions occur more frequently in the acute than in the chronic form of the disease.

Chronic regional enteritis which is intractable to medical treatment or is complicated by obstruction, bleeding, or fistula formation should be treated by resection of the involved bowel. The mortality rate for surgical treatment ranges from 2-14%. In recent years surgical mortality rates have been generally low, chiefly due to improved preoperative and postoperative care. It is now considered safe to resect involved bowel rather than to perform simple short-circuiting procedures such as ileocolostomy. The recurrence rate after surgery is 20-60%.

NEOPLASMS OF SMALL BOWEL

Benign Small Bowel Tumors.

These include lipomas, leiomyomas, fibromas, benign carcinoids, polyps, and hemangiomas. Growth is very slow. These tumors produce symptoms by bleeding, obstruction, or by functioning as the leading point of an intussusception. Surgical excision is indicated. Islands of aberrant pancreatic tissue may occur in the bowel wall and are grossly indistinguishable from neoplasms.

Gastrointestinal polyposis associated with melanin spots on the oral mucosa, lips, and digits is a rare but important familial condition (Peutz-Jeghers syndrome). The diagnosis can be readily inferred from the characteristic pigmentation. The polyps in this syndrome rarely become malignant and do not require removal unless they cause a complication such as intussusception or bleeding.

Malignant Small Bowel Neoplasms.

The most important of these are adenocarcinoma, lymphosarcoma, and malignant carcinoid. Small bowel malignancies tend to occur in somewhat younger age groups than gastric and colon carcinomas. Initial symptoms are often vague and misleading, consisting of intermittent abdominal pain or colic suggestive of partial obstruction. Vomiting may occur, and melena is occasionally present. An abdominal mass may be palpable. Acute intussusception may develop. Early diagnosis depends upon a high index of suspicion and insistence on complete and careful x-ray study of the entire small bowel.

Treatment is by resection. Postoperative radiotherapy is of value in lymphosarcoma but in none of the other tumors.

Carcinoid occurs in any part of the gastrointestinal tract, most commonly in the appendix where its behavior is usually benign. The next highest incidence is in the small bowel. Malignant carcinoid with hepatic metastases may produce a syndrome consisting of a peculiar flushing and cyanosis, transient patchy skin lesions, diarrhea, dyspnea, and right-sided cardiac valvular disease. These clinical developments are caused by an excess of serotonin (5-hydroxytryptamine) secreted by the tumor, combined with a failure of the normal detoxification of the hormone by the involved liver. Examination of the urine for 5-hydroxyindoleacetic acid (a serotonin end-product excreted by the kidneys) should be performed in suspicious cases. The test may also be of value as an index of recurrence during the follow-up after resection of malignant carcinoid.

DISEASES OF THE COLON
AND RECTUM

Diagnostic Procedures.

In addition to the routine work-up (see p. 259), patients with significant large bowel symptoms should be investigated by means of 1 or more of the following procedures: (1) Examination of the stools for occult blood, ova, and parasites, and culture for enteric pathogens; (2) anoscopy; (3) sigmoidoscopy with smear, culture, or

biopsy of suspicious lesions; (4) barium enema with or without air contrast study.

Preoperative Preparation.

A. General Measures: When a difficult exposure at operation or an unusual degree of postoperative ileus is anticipated, a long intestinal tube should be passed well into the ileum preoperatively. In less complicated cases a gastric tube is inserted on the morning of colon surgery. If the operative field in the pelvis will be obscured by a full bladder, or if urinary retention is to be expected postoperatively (as after a combined abdominoperineal resection), a Foley catheter should be inserted before surgery and placed on constant drainage.

B. Bowel Preparation: Preparation of the colon with antibacterial agents is advisable prior to elective abdominal operations during which the large intestine may be opened.

   1. Principles -

      a. Adequate doses of nonabsorbable bactericidal drugs must be administered. The course should be as short and intensive as possible, because prolonged reduction of the normal bacterial population of the intestines permits overgrowth of yeasts or of resistant pathogens such as Staphylococcus aureus (which may cause serious staphylococcal enterocolitis).

      b. There must be no significant intestinal obstruction.

      c. Diet - A minimal-residue diet is instituted just prior to beginning drug therapy.

      d. Cleansing of the bowel with enemas or cathartics (see p. 614) should precede oral administration of an antibacterial agent.

   2. Specific regimens - Neomycin is the most effective single antibiotic agent. Its antibacterial effect may be increased slightly by the addition of other agents.

      a. Twenty-four hour preparation (Poth) -

        ℞ Neomycin   1 Gm.
          Phthalylsulfathiazole (Sulfathalidine®) 1.5 Gm.
            Sig.: At 2:00, 3:00, 4:00, 8:00, and 12:00 p.m., and at 4:00 and 8:00 a.m.
        (Operation at 9:00 a.m.)

      b. Thirty-six hour preparation (Dearing) -

        ℞ Neomycin   1.5 Gm.
          One of the tetracyclines   0.25 Gm.
            Sig.: Every hour for 4 doses the first afternoon, then 4 times on the second day.
        (Operation on the following morning.)

      c. Three-day preparation (Fog) -

        ℞ Neomycin   1 Gm.
          Bacitracin   1 Gm. (120,000 units)
            Sig.: T.i.d. for 3 days.
        (Operation on the fourth morning.)

If neomycin is unavailable, the bowel may be prepared with succinylsulfathiazole (Sulfasuxidine®) or phthalylsulfathiazole (Sulfathalidine®), 5 Gm. (75 gr.) q.i.d. for 5-7 days.

As a precaution against the postoperative invasion of the colon by Staphylococcus aureus, bacitracin, 10,000 units orally 4 times a day for 3 days after operation, may be given as soon as the patient can tolerate fluids by mouth.

DIVERTICULOSIS AND DIVERTICULITIS

Diverticulosis is rare in persons under 40 years of age, but the incidence increases with age and it is probable that more than half of people over 80 have diverticula. The lesions are confined to the sigmoid in 40% of cases. Only a very small proportion of those with diverticulosis develop diverticulitis. The site of diverticulitis is in the sigmoid in about 90% of cases.

Clinical Findings.
The major symptoms of chronic diverticulitis are changes in bowel habits (constipation, diarrhea, or flatulence), recurrent left lower quadrant pain and tenderness, and, occasionally, rectal bleeding. Acute sigmoid diverticulitis is characterized by the rapid onset of severe left lower quadrant pain, tenderness, and signs of peritoneal irritation. Fever and leukocytosis are usually present. An inflammatory mass is frequently palpable in the left lower quadrant.

Definitive diagnosis can usually be made by barium enema. Differentiation from carcinoma is occasionally difficult or impossible. Coexistent cancer and diverticulosis may produce a confusing clinical picture. Sigmoidoscopy should always be done, even though the inflammatory changes of diverticulitis are usually above the reach of the sigmoidoscope.

Treatment.
A. Medical Measures:
 1. Chronic - About 75% of patients with chronic diverticulitis will improve on low-residue diet, anticholinergic drugs, and control of constipation.
 2. Acute diverticulitis is treated as for acute peritonitis (see p. 260). It is important to give combined antibiotic therapy with penicillin and streptomycin and (in severe cases) chloramphenicol or 1 of the tetracyclines (see p. 614). Most attacks can be controlled by conservative measures. Surgery during an acute episode should be avoided if possible.
B. Surgical Treatment: Resection is indicated if any of the following occur: (1) perforation; (2) fistula formation to the bladder, bowel, or elsewhere; (3) obstruction; (4) massive hemorrhage; and (5) chronic or recurrent acute symptoms. If there are no contraindications, resection is advisable for patients who have persistent annoying symptoms or more than 1 acute attack. This policy will lower the incidence of serious complications of the disease, which may require complex and staged procedures.

When possible, operation should be deferred until the local inflammatory reaction has subsided on medical management. This "cooling off" period usually requires 3-6 months after a severe acute attack.

Emergency operation (because of persistent local sepsis) during an acute episode should usually be limited to the establishment of a diverting transverse colostomy and drainage of abscesses if present.

If the disease has reached an advanced stage, with fistula formation or severe local induration and scarring, a transverse colostomy is necessary so that the distal bowel can remain at rest for several months before resection is attempted. The necessity for staged operations has diminished in recent years.

Prognosis.

When local inflammation has subsided, colon resection with primary anastomosis can be accomplished with a mortality rate of about 1%. Patients usually remain asymptomatic after an adequate resection for diverticulitis.

ULCERATIVE COLITIS

Clinical Findings.

Ulcerative colitis usually begins between 20 and 30 years of age. The cause is not known. Onset is commonly with diarrhea (often bloody), fever, and weight loss. The initial attack may be mild or severely fulminant; the disease often becomes chronic, with recurrent periods of diarrhea and prostration which lead to semi-invalidism and such complications as colonic perforation, cirrhosis, arthritis, perianal fistulas, malnutrition, or carcinoma. Cancer of the colon develops in 10-30% of cases of over 10 years' duration.

Diagnosis is usually apparent on sigmoidoscopy because the disease involves the upper rectum in about 95% of cases. On sigmoidoscopy in early cases, the mucosa appears edematous and hyperemic, and bleeds easily; irregular superficial ulcerations develop later. In the early stages, barium enema reveals typical ulcerations. Shortening of the colon, loss of haustration, and tubular configurations are signs of advanced disease.

In the differential diagnosis of ulcerative colitis, other causes of diarrhea must often be considered, including amebiasis, dysentery, tuberculous enteritis, sprue, and regional enteritis.

A severe fulminating form of ulcerative colitis is seen occasionally. It is characterized by abdominal pain, distention, severe bloody diarrhea, prostration, fever, leukocytosis, and marked dehydration. Death may occur from peritoneal sepsis, hemorrhage, or overwhelming toxemia. Because of the danger of colonic perforation, these patients must be observed carefully for an acute abdomen.

Treatment.
  A. Medical Measures:
    1. Severe, fulminating colitis - Treatment of this acute, necrotizing infection consists of appropriate parenteral fluid therapy (see p. 99), systemic combined antibiotic therapy

(e.g., penicillin and a tetracycline; see p. 614), blood transfusions, and sedation (see p. 607). When severe systemic symptoms persist, corticotropin or cortisone may give dramatic relief (see p. 607). **Caution:** These drugs may mask symptoms of perforation or sepsis.

Emergency ileostomy is rarely necessary.

2. Chronic, debilitating colitis - These patients should have a bland diet from which all foods discovered to aggravate the diarrhea have been eliminated. Medications should include anticholinergics, mild sedatives, and nonnarcotic antidiarrheal drugs. Oral nonabsorbable antibacterial agents may be of temporary benefit. When fever and systemic signs of sepsis are present, parenteral combined antibiotic therapy is indicated (see above).

B. Surgical Treatment:

1. Indications - Surgery should be advised only after a thorough trial of medical management, but must not be delayed until the patient is terminal or debilitated. Operation is indicated if the patient is disabled by recurrent attacks, malnutrition, or impairment of general health. Complications such as arthritis, ulcerative dermatitis, hepatitis, nephritis, acute and subacute colonic perforation, colonic obstruction, hemorrhage, and intractable perianal infection are additional indications for surgery. Because chronic ulceration is a precursor of a highly malignant type of cancer, severely diseased bowel should be removed. Until this can be done, barium enema and sigmoidoscopy should be carried out every 6 months. Symptoms suggestive of malignancy (e.g., rectal bleeding, abdominal pain, or change in bowel habits) are often ignored by these patients, who have usually had such complaints for years.

2. Preoperative preparation - Intensive medical treatment as outlined above is employed. Significant blood and protein losses due to diarrhea commonly occur and require repeated transfusions of whole blood. Normal Hgb. and plasma protein levels should be restored before operation if possible. In severe cases corticotropin or cortisone (see p. 607) is administered preoperatively for as long as required (usually 1 to several weeks) to induce a temporary remission. If corticosteroid therapy is instituted, it must be administered after operation and discontinued gradually.

3. Operations for ulcerative colitis - Ileostomy alone may be indicated as a life-saving measure in fulminating disease. Usually the procedure of choice is ileostomy with subtotal colectomy or, if the patient's condition permits, ileostomy with proctocolectomy in 1 stage. There is no loss of sexual function if the dissection is kept close to the rectum. The entire colon and rectum should be removed eventually except in selected patients with limited localized involvement of the colon; in this small group, segmental resection of the colon may be adequate.

Prognosis.

Medical management in refractory ulcerative colitis has a mortality rate of 10-20%, and the same group of patients can now

be operated on with a mortality of less than 5%. Patients with ileostomies can lead normal lives and, if the entire colon and rectum have been removed, are usually cured.

Complications of Ileostomy.

A. Skin Irritation: This is the most common complication. Its prevention depends upon constant protection of the skin by a properly fitting ileostomy appliance. A plastic bag is cemented to the skin around the ileostomy at the operating table and changed as often as necessary during the following week to prevent contact of ileal contents with the skin. A permanent device is then used. If skin excoriation develops, the same measures may be used as in small bowel fistula (see p. 291).

B. Excessive Fluid Loss: Copious fluid stools may occur from the ileostomy immediately postoperatively or intermittently at any time thereafter. Patients should be warned of this possibility and cautioned to seek hospitalization if fluid loss is marked. Prompt correction of electrolyte imbalance is essential. Partial obstruction of the ileostomy or the ileum should be suspected. Insertion of a well lubricated gloved finger or soft rectal tube into the ileostomy may relieve a temporary local obstruction or dysfunction. Antidiarrheal agents and low-residue diet are usually indicated.

C. Prolapse: This is usually a late complication related to inadequate internal fixation of the ileum or to relaxation of the abdominal ring. If prolapse is recurrent and severe, a new ileostomy should be made; excision of the prolapsed ileum will not cure the condition. Wound herniation, when marked, also requires a new ileostomy.

D. Stricture and Fistula at the Skin Level: These may be early or late complications. They are due to errors in surgical technic or to chronic infection, and require local revision of the ileostomy.

E. Mechanical Obstruction: Small bowel obstruction may be an early or late complication. Surgical intervention as for other forms of small bowel obstruction may be necessary (see p. 267).

ADENOMA (ADENOMATOUS POLYPS)
OF COLON AND RECTUM*

Adenomatous polyps are found in the colon in about 9% of autopsies. Two-thirds are in the sigmoid; the remainder are scattered elsewhere. Whether polyps are precancerous lesions is an unsettled question. Malignant change is seen occasionally in the superficial cells, but invasion of the basement membrane and base of the polyp is rare.

Clinical Findings.

Painless, slight rectal bleeding is the commonest symptom, but most polyps are asymptomatic. Sigmoidoscopy and double contrast barium enema are done on all patients suspected of having a

*Rare benign neoplasms of the colon and rectum (lipoma, angioma, fibroma, leiomyoma, carcinoid) will not be discussed.

polyp. The colon should always be studied by x-ray when polyps
are found in the rectum. Before laparotomy for adenoma in the
colon, the double contrast barium enema should usually be repeated
to verify the original findings.

Treatment.
Adenomas of the colon and rectum should be completely re-
moved and examined microscopically; fragments of the lesion may
not be representative. If the lesion is benign or if noninvasive
malignancy is present, complete local excision is adequate. In-
vasive carcinoma (as indicated by involvement of the base of the
adenoma) must be treated as outlined for carcinoma (see p. 300).
A. Adenomas Within Reach of the Sigmoidoscope:
   1. Very small adenomas or mucosal excrescences are com-
      pletely excised with the cold biopsy forceps and the base
      fulgurated with diathermy.
   2. Pedunculated adenomas are held under tension with grasping
      forceps while the base is coagulated with the diathermy.
      The base is then divided with a diathermy snare or with a
      biopsy forceps and coagulated.
   3. Sessile adenomas (smooth and villous) can be removed with
      an electric snare or a double-loop diathermy resector. Co-
      agulation and piecemeal removal of small lesions of this
      type is occasionally justifiable if multiple biopsies with the
      cold forceps show no evidence of malignancy. When sessile
      adenomas can be delivered to a sufficiently low level for a
      submucosal surgical excision and closure with 000 chromic
      catgut, this is the preferred method of treatment. The in-
      cidence of invasive cancer is higher in the villous type of
      adenoma.
B. Adenomas Above the Reach of the Sigmoidoscope: All of these
   lesions require laparotomy after bowel preparation with an
   intestinal antiseptic (see p. 294). Colon polyps are usually
   excised locally through a colotomy. If cancer is suspected,
   frozen sections are obtained, but the choice of treatment can
   usually be based on the gross appearance of the polyp. Be-
   cause polyps are frequently multiple, careful palpation of the
   entire colon at operation is essential. The surgeon may wish
   also to pass a sterile sigmoidoscope through colotomies for
   examination of the interior of the bowel.

Follow-up.
Pedunculated polyps rarely recur, but sessile lesions not un-
commonly do so after local removal. All patients with adenoma
within reach of the sigmoidoscope should be followed by endoscopy
every 2 months for the first 6 months and every 6-12 months there-
after. Examination with double contrast barium enemas every
12-18 months is advisable after removal of a colon polyp.

FAMILIAL INTESTINAL POLYPOSIS

This is a rare hereditary disease characterized by innumerable
adenomatous polyps in the colon and rectum. Patients with this
disorder usually develop cancer of the large bowel, sometimes at a

very early age. The condition is transmitted as a heterozygous dominant mendelian trait. The following are additional important features: (1) Males and females are affected equally, and both may transmit the disease. (2) It is probable that only those who have inherited polyposis can transmit it. (3) In polyposis families, usually only one-half of the children inherit the disease. (4) The severity of the disease and the tendency to develop cancer of the colon or rectum vary considerably in different families. (5) When polyposis develops early in life, cancer frequently occurs within 10-15 years; whereas when polyposis develops later the precancerous incubation period is longer. All patients with polyposis do not give a family history of the disease, but the danger of malignancy is the same in these sporadic cases.

Clinical Findings.

Diarrhea and bleeding from the bowel are the 2 most common symptoms. The diagnosis can always be made by sigmoidoscopy and the extent of involvement determined by double contrast barium enema. A negative barium enema, however, does not exclude the presence of multiple small sessile polyps in the colon. A careful family history should always be taken and other members of the family, who may be asymptomatic, advised to have an examination. The only disorder likely to be confused with polyposis is the pseudo-polyposis of ulcerative colitis, but the differentiation can usually be made by history, sigmoidoscopy, and biopsy.

Treatment.

Colectomy (with preservation of only the distal 15 cm. of the rectum) and end-to-end ileoproctostomy is the treatment of choice. Residual rectal polyps can be removed by excision and electrocoagulation through the sigmoidoscope. Such patients require lifelong follow-up at intervals of 6 months.

Evidence of carcinoma on biopsy of a rectal polyp is an indication for combined abdominoperineal resection. When a patient cannot be adequately followed, total proctocolectomy and ileostomy should be done initially.

Adenomas in the rectal stump have been observed to disappear or regress spontaneously after total colectomy and low ileorectal anastomosis.

CARCINOMA OF COLON AND RECTUM

Carcinoma is the only common malignancy of the colon and rectum. Lymphoma, malignant carcinoid, melanoma, fibrosarcoma, and other types of sarcoma do occur but are very rare. The treatment of all is essentially the same.

Carcinoma of the colon and rectum causes more deaths than any other form of cancer. The only known predisposing causes are familial multiple polyposis, chronic ulcerative colitis, chronic lymphogranuloma venereum, chronic granuloma inguinale, and perhaps adenoma. Males are affected more commonly than females in a ratio of 3:2. The highest incidence is in patients about 50 years of age, but occasional cases have been reported in younger persons and even in children. The anatomic distribution of cancer of the large bowel (based on a study of about 5000 cases) is approximately

16% in the cecum and ascending colon, 5% in the transverse colon, 9% in the descending colon, 20% in the sigmoid, and 50% in the rectum.

One-half to two-thirds of all lesions of the colon and rectum lie within reach of the examining finger or sigmoidoscope and therefore can be biopsied on the first visit.

Clinical Findings.

Symptoms vary depending upon whether the lesion is in the right or the left side of the colon. An acute abdominal emergency may be precipitated by perforation or intussusception. The definitive diagnostic procedures in all cases are sigmoidoscopy and barium enema.

A. Carcinoma of the Right Colon: Because the fecal stream is fluid and the bowel lumen large in the right half of the colon, symptoms of obstruction occur less frequently than in left-sided tumors. Flatulence is often the only initial complaint. This may progress to cramplike pain, occasionally simulating cholecystitis or appendicitis. Secondary anemia with associated weakness and weight loss is found in half of patients with right colon lesions. The stools are usually positive for occult blood, but rarely show gross blood. The patient is likely to have diarrhea. The first indication of cancer may be the discovery of a palpable mass in the right lower quadrant.

B. Carcinoma of the Left Colon: Obstructive symptoms predominate, particularly increasing constipation. There may be short bouts of diarrhea. Occasionally the first sign is acute colonic obstruction. A small amount of bright red bleeding with bowel movements is common, and anemia is found in about 20% of cases. At times a mass is palpable. About half of cases give a history of weight loss.

Treatment.

The only curative treatment in cancer of the large bowel is wide surgical resection of the lesion and its regional lymphatics after adequate bowel preparation (see p. 294) and appropriate supportive measures. When a significant degree of mechanical obstruction is present, a preliminary transverse colostomy or cecostomy is necessary. Even though the lesion is incurable, palliative resection may be of value to relieve obstruction, bleeding, or the symptoms of local invasion.

Management of the Bladder After Combined Abdominoperineal Resection.

Postoperative urinary retention occurs in one-fourth of abdominoperineal resections and persists longer than 3 months in 10% of cases. Formerly this was regarded as neurogenic, but present opinion holds that mechanical factors are largely responsible. Some degree of prostatism is frequently present, and is aggravated by loss of support of the base of the bladder, vesical neck, and prostatic urethra, so that the physiologic balance is disturbed and the bladder decompensates.

Constant bladder drainage with a Foley catheter is maintained for 7 days after a combined abdominoperineal resection. If by this time the patient is fully ambulatory and convalescence is normal,

the catheter is removed in the morning and voiding is attempted. The amount of residual urine is determined that afternoon or evening. If more than 150 ml. are present, either the catheter is replaced for 48 hours or the patient is catheterized several times at 8-10 hour intervals. Even if voiding seems satisfactory, the patient should be catheterized for residual urine once daily for 2-3 days. If voiding is poor, Bethanechol Chloride, U.S.P. (Urecholine®), may be helpful. In patients whose general condition and convalescence are satisfactory, ineffective conservative treatment should not be prolonged more than 3 weeks. Transurethral resection of the prostate is then done with immediate excellent results in about 90% of patients.

Care of the Colostomy.

The commonest permanent colostomy is the sigmoid colostomy made at the time of combined abdominoperineal resection. Abdominal distention must be avoided postoperatively by gastric tube suction until bowel activity returns. This is essential because tension on the colostomy involves the danger of retraction.

Colostomy irrigation is begun about 1 week after operation. Each day, a well-lubricated catheter or rectal tube is gently inserted about 15 cm. (6 in.) into the colostomy and 500-1000 ml. of water are instilled from an enema can or bag held 30-60 cm. (1-2 feet) above the colostomy. After the bowel has become accustomed to regular enemas, evacuation will occur within about one-half hour after the irrigation. Some individuals have regular movements without irrigation. A small gauze or disposable tissue pad worn over the colostomy, held in place by a wide elastic belt or ordinary girdle, is usually all the protection required during the day. For several months postoperatively the patient dilates the colostomy once daily by insertion of an index finger. Commercial colostomy kits make care simple and convenient.

Three important principles of colostomy management are routine time for bowel evacuation; complete emptying after irrigation; and regulation of diet to avoid diarrhea. The patient with a colostomy can live a normal life.

Stricture, prolapse, and wound hernia are late colostomy complications requiring surgical correction. Skin irritation is less likely to occur than with ileostomy.

Prognosis.

Over 90% of patients with carcinoma of the colon and rectum are suitable for either curative or palliative resection, with an operative mortality of 3-6%. The over-all five-year survival rate after resection is about 50%. If the lesion is confined to the bowel and there is no evidence of lymphatic or blood vessel invasion, the five-year survival rate is 60-70%. Local recurrence of carcinoma in the anastomotic suture line or wound area occurs in 10-15% of cases. The incidence of local recurrence can be decreased if special precautions are taken at operation to avoid implantation of malignant cells. About 5% of patients develop multiple primary colon cancers. Early identification of resectable local recurrence or a new neoplasm depends upon careful follow-up with sigmoidoscopy and barium enema every 6 months for 2 years and yearly thereafter.

AMEBIASIS OF THE COLON

Amebiasis of the colon is of surgical significance chiefly because it sometimes mimics other conditions. Fulminating amebic colitis may resemble ulcerative colitis and may result in perforation (usually of the cecum) or hemorrhage. Amebic granuloma or "ameboma" is a complication of chronic amebiasis and involves any part of the colon, but particularly the cecum or rectum. Differentiation from carcinoma may be difficult.

The diagnosis is established by sigmoidoscopic and stool examinations. Acute amebiasis is usually associated with superficial, ragged ulcerations in the upper rectum within reach of the sigmoidoscope. Motile amebas are found on fresh smears from these ulcers. In chronic intestinal amebiasis, amebic cysts are present in the stools but may be difficult to find.

Treatment is with antiamebic drugs. Surgical measures may rarely be required in the management of complications.

RECTAL PROLAPSE

Partial or incomplete rectal prolapse consists of eversion of the rectal mucosa. It can be differentiated from hemorrhoids by the fact that the ridges of mucosa are arranged concentrically instead of radially, as is characteristic of hemorrhoidal prolapse. In complete prolapse, all layers of the rectal wall are involved and a considerable segment of bowel may be everted through the relaxed anal canal. Concealed prolapse consists of an intussusception of the rectum within the rectum and without protrusion through the anus.

Prolapse occurs most commonly in young children or in persons past middle age. The underlying cause is a weakness in the supporting structures. The diagnosis is usually obvious. In suspected concealed prolapse, it is advisable to do a rectal examination with the patient standing because the intussusception may be reduced in the horizontal position.

Conservative treatment is often successful in young children. The patient should lie down to defecate, and the buttocks should be firmly strapped together at other times. Partial prolapse in older patients may respond to multiple submucosal injections of quinine and urea hydrochloride (5% solution); this should be attempted only by one experienced with the method. If this fails, the partial prolapse may be amputated by the Buie technic. Complete prolapse can be treated successfully by a variety of operations; 1 method consists of freeing the rectum from the hollow of the sacrum, suturing it high to supporting structures to obliterate the cul-de-sac, and resecting redundant bowel via the abdomen. An alternative procedure is resection of the prolapse via the perineal approach.

DISEASES OF THE ANAL CANAL

HEMORRHOIDS

Internal hemorrhoids are varices of that portion of the venous hemorrhoidal plexus which lies submucosally just proximal to the dentate margin. External hemorrhoids arise from the same plexus but are located subcutaneously immediately distal to the dentate margin. There are 3 primary internal hemorrhoidal masses: right anterior, right posterior, and left lateral. Three to 5 secondary hemorrhoids may be present between the 3 primaries. Portal obstruction and pregnancy are important specific causes of hemorrhoids, but in most cases the etiology is obscure. Straining at stool, constipation, prolonged sitting, and anal infection are contributing factors and may precipitate complications such as thrombosis. Diagnosis is suspected on the history of protrusion, anal pain, and bleeding and confirmed by proctologic examination.

Carcinoma of the colon or rectum not infrequently aggravates hemorrhoids or produces similar complaints. For this reason, surgical treatment of hemorrhoids is always preceded by sigmoidoscopy and barium enema. Mild internal hemorrhoids frequently improve spontaneously or on regulation of bowel habits with mineral oil or other nonirritating laxatives (see p. 610). For more severe involvement, complete internal and external hemorrhoidectomy in the hospital is a highly satisfactory procedure when properly done. Excision of a single external hemorrhoid, evacuation of a thrombosed pile, and the injection treatment of internal hemorrhoids fall within the scope of office practice.

Thrombosed external hemorrhoids result from the rupture of a vein at the anal margin, forming a clot in the subcutaneous tissue. The patient complains of a painful lump, and examination shows a tense, tender, bluish mass covered with skin. If seen after 24-48 hours when the pain is subsiding - or if symptoms are minimal - hot sitz baths are prescribed. If discomfort is marked, removal of the clot is indicated. With the patient in the lateral position, the area is prepared with antiseptic and 1% procaine is injected intracutaneously around and over the lump. A radial ellipse of skin is then excised and the clot evacuated. A dry gauze dressing is held in place for 12-24 hours by taping the buttocks together, and daily sitz baths are then begun.

Injection Treatment of Internal Hemorrhoids.

This method is most suitable for small internal hemorrhoids which bleed or for palliation of large hemorrhoids in patients who are unable to undergo operation. Injection therapy should never be used on external hemorrhoids or in the presence of infection. Quinine and urea hydrochloride (5% solution) or sodium morrhuate (5% solution) may be used as the sclerosing agent.

Technic: With the patient in the left lateral position the internal hemorrhoid is visualized with the anoscope. A few drops of sclerosing solution are injected through a No. 20 gauge needle into the center of each internal hemorrhoid under direct vision. Care is taken to make the injection above the mucocutaneous line, as injection or seepage of the solution beneath the skin causes severe

pain. If the injection is made too superficially, the mucosa will blanch and necrosis may follow. Each internal hemorrhoid is treated at each visit; a total dose of 1 ml. is rarely exceeded on any single occasion.

The actual injection is practically painless, although mild pain may begin in about 30 minutes and last for an hour or so; this can be relieved by heat or lying down. Symptoms are often relieved by the first treatment, but maximum benefit will often require 5-6 treatments at intervals of 1-2 weeks. Bleeding can be controlled in 90% of cases, and protrusion is relieved in over half. Complications are minimal if the proper technic is used, and drug reactions are rare. Some induration can often be felt in the hemorrhoid for a few days after treatment; further injections should be delayed until this has subsided, or the injection site may slough.

CRYPTITIS AND PAPILLITIS

Anal pain and burning of brief duration with defecation is suggestive of cryptitis and papillitis. Digital and anoscopic examination reveals hypertrophied papillae and indurated or inflamed crypts. Treatment consists of mineral oil by mouth (see p. 610); anorectal ointment (Nuzine®) or suppository (Anusol®) after each bowel movement; and local application of 5% phenol in oil or carbolfuchsin compound to the crypts. If these measures fail, surgical excision of involved crypts and papillae should be considered.

FISSURE IN ANO

Acute fissures represent recent breaks in the anal lining caused by the trauma of bowel movements. They usually clear if bowel movements are kept regular and soft (e.g., with mineral oil). The local application of 1% gentian violet solution or a mild styptic such as 1-2% silver nitrate may be of value.

Chronic fissure is characterized by (1) acute pain during and after defecation; (2) spotting of bright red blood at stool with occasional more abundant bleeding; (3) tendency to constipation through fear of pain; and (4) the late occurrence of a sentinel pile, hypertrophied papilla, and spasm of the anal canal (usually very painful on digital examination). Regulation of bowel habits with mineral oil or other stool softeners (see p. 610), sitz baths, and anal suppositories (e.g., Anusol®), b.i.d., should be tried. If these measures fail, the fissure, sentinel pile, or papilla and the adjacent crypt must be excised surgically. Postoperative care is along the lines of the preoperative treatment.

ANAL ABSCESS

A perianal abscess should be considered the acute stage of an anal fistula until proved otherwise. The abscess should be adequately drained as soon as it has localized. Hot sitz baths may hasten the process of localization. The patient should be warned that after drainage of the abscess he may have a persistent fistula. It

Papillitis

Cryptitis

Pectinate line

Thrombotic hemorrhoid

Anal carcinoma

Prolapsing hemorrhoid

Fistula

Abscess

Fissure in ano
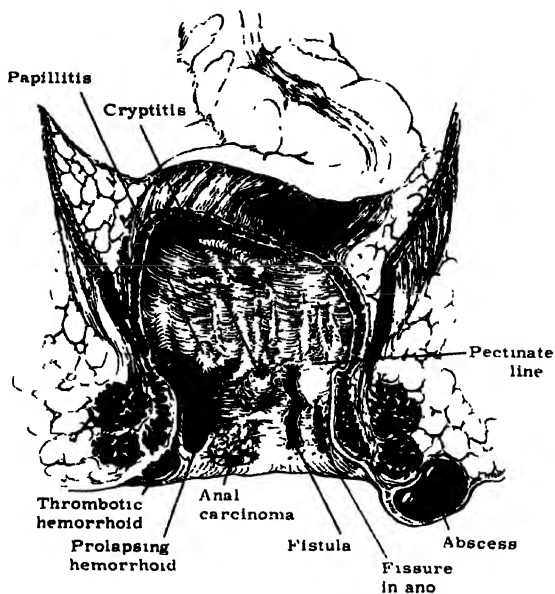
Common Lesions of the Anal Canal

Levator ani muscle

Deep external sphincter muscle

Internal sphincter muscle

Superficial external sphincter muscle

Subcutaneous sphincter muscle
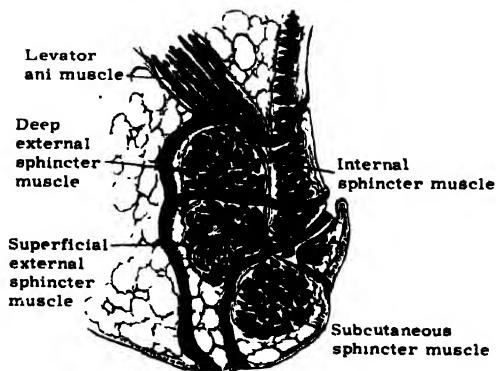
Cross-section of Muscles of Anal Wall Showing Usual Paths of Anal Fistulas

is painful and fruitless to search for the internal opening of a fistula in the presence of acute infection.

FISTULA IN ANO

About 95% of all anal fistulas arise in an anal crypt and they are often preceded by an anal abscess. If an anal fistula enters the rectum above the pectinate line and there is no associated disease in the crypts, ulcerative colitis, rectal tuberculosis, lymphogranuloma venereum, cancer, or foreign body should be considered in the differential diagnosis.

Anal fistula is associated with the chronic purulent discharge from the fistulous opening on the skin near the anus. There is usually local itching, tenderness, or pain aggravated by bowel movements. Recurrent anal abscesses may develop. The involved crypt can occasionally be located anoscopically with a crypt hook. Probing the fistula should be gentle because false passages can be made with ease, and in any case demonstration of the internal opening by probing is not essential to the diagnosis.

Treatment is by surgical incision or excision of the fistula under general anesthesia. If a fistula passes deep to the entire anorectal ring so that all the muscles must be divided in order to extirpate the tract, a two-stage operation must be done to prevent incontinence.

PRURITUS ANI

Among the common causes of anal itching are intertrigo, discharges from fistulas, folliculitis, fungal infection, including the common tinea cruris and monilial dermatitis (secondary to administration of broad-spectrum antibiotics); drug eruption, contact dermatitis from local applications, primary dermatosis such as psoriasis or seborrheic dermatitis; parasitic infestation such as pediculosis pubis, scabies, and enterobiasis (pinworms); and psychogenic causes.

Treatment should be specific whenever possible, but symptomatic therapy is usually necessary. Sensitizing agents (e.g., anesthetics and antihistamines) should be avoided. When the skin is hot and inflamed, ice-cold normal saline dressings and calamine liniment are applied. If there is only mild skin inflammation, simple calamine lotion with 0.25-0.5% menthol and 0.5-1% phenol may be used; or a bland application such as Hydrophilic Ointment, U.S.P., with 0.25-0.5% menthol and 0.5-1% phenol. Hydrocortisone, ointment is often very effective. In severe cases (rare), oral or parenteral cortisone therapy may be indicated (see p. 607). Sedatives and soporifics may be required (see p. 607). Local cleanliness, talc applications to promote dryness, and avoidance of sweating and chafing are helpful. Do not use x-ray therapy or local injections of anesthetic agents. The only indications for surgical treatment are local lesions such as hemorrhoids, fistulas, and fissures which may be contributing to the pruritus.

ANAL CONDYLOMAS

These wart-like papillomas of the perianal skin and anal canal flourish on moist, macerated surfaces, particularly in the presence of purulent discharge. They are not true tumors but are infectious and auto-inoculable, probably due to a virus. They must be distinguished from condyloma lata caused by syphilis. The diagnosis of the latter rests on the positive serologic test for syphilis or the discovery of Treponema pallidum on dark-field examination.

Treatment consists of careful application of 25% podophyllin in tincture of benzoin to the lesion (with bare wooden or cotton-tipped applicator sticks to avoid contact with uninvolved skin). Condylomas in the anal canal are treated through the anoscope and the painted site dusted with powder to localize the application and minimize discomfort. Electrofulguration under local anesthesia is useful if there are numerous lesions. Local cleanliness and the frequent use of a talc dusting powder are essential.

Condylomas tend to recur. The patient should be observed for several months and advised to report promptly if new lesions appear.

BENIGN ANORECTAL STRICTURES

Congenital.

Anal contracture or stenosis in infancy may result from failure of disintegration of the anal plate in fetal life. The narrowing is treated by careful repeated dilatation, inserting progressively larger Hegar dilators until the anus admits first the little and then the index finger.

Traumatic.

Acquired stenosis is usually the result of surgery or trauma which denudes the epithelium of the anal canal. Hemorrhoid operations in which too much skin is removed or which are followed by infection are the commonest cause. Constipation, ribbon stools, and pain on defecation are the most frequent complaints. Stenosis predisposes to fissure, low-grade infection, and occasionally fistula.

Prevention of stenosis after radical anal surgery is best accomplished by local cleanliness, hot sitz baths, and gentle insertion of the well-lubricated finger twice weekly for 2-3 weeks beginning 2 weeks after surgery. When stenosis is chronic but mild, graduated anal dilators of increasing size may be inserted daily by the patient. For marked stenosis a plastic operation on the anal canal is advisable.

Inflammatory.

A. Lymphogranuloma Venereum: This viral disease is the commonest cause of inflammatory stricture of the anorectal region. Acute proctitis due to lymphatic spread of the virus occurs early, and may be followed by perirectal infections, sinuses, and formation of scar tissue (resulting in stricture). Frei and complement fixation tests are positive.

The tetracycline drugs are curative in the initial phase of the disease. When extensive chronic secondary infection is

present or when a stricture has formed, repeated biopsies are essential because epidermoid carcinoma develops in about 4% of strictures. Local operation on a stricture may be feasible, but a colostomy or an abdominoperineal resection is often required.

B. Granuloma Inguinale: This disease may cause anorectal fistulas, infections, and strictures. The Donovan body is best identified in tissue biopsy when there is rectal involvement. Epidermoid carcinoma develops in about 4% of cases with chronic anorectal granuloma.

The early lesions respond to tetracyclines. Destructive or constricting processes may require colostomy or resection.

ANAL INCONTINENCE

Obstetric tears, anorectal operations (particularly fistulotomy), and neurologic disturbances are the most frequent causes of anal incontinence. When incontinence is due to surgery or trauma, surgical repair of the divided or torn sphincter is indicated. Repair of anterior laceration due to childbirth should be delayed for 6 months or more after parturition.

SQUAMOUS CELL CARCINOMA OF THE ANUS

These tumors are relatively rare, comprising only 1-2% of all malignancies of the anus and large intestine. Bleeding, pain, and local tumor are the commonest symptoms. Because the lesion is often confused with hemorrhoids or other common anal disorders, immediate biopsy of any suspicious mass or ulceration in the anal area is an essential diagnostic precaution. These tumors tend to become annular, invade the sphincter, and spread upward into the rectum.

Except for very small lesions (which can be adequately excised locally), treatment is by combined abdominoperineal resection. Radiation therapy is reserved for palliation and for patients who refuse or cannot withstand operation. Metastases to the inguinal nodes are treated by radical groin dissection when clinically evident. The five-year survival rate after resection is about 50%.

DISEASES OF THE
BILIARY SYSTEM AND LIVER

CHOLELITHIASIS

The high incidence of gallstones in the general population accounts for the clinical frequency of cholecystitis. Autopsy studies show that 32% of women and 16% of men past the age of 40 have gallstones. The incidence of calculi rises sharply at around 40 years of age. Pregnancy is an important predisposing cause of gallstones, and obesity may also be a contributing factor; hence

the description of the typical gallbladder patient as "female, fat, and forty."

Gallstones usually consist of cholesterol, calcium bilirubinate, calcium carbonate, or a mixture of these. About 90% of the stones associated with chronic cholecystitis are of the mixed variety, whereas the preceding 3 types of "pure" calculi may be seen in a relatively normal gallbladder. Calcium bilirubinate stones tend to occur, sometimes at an early age, in such diseases as congenital hemolytic anemia and sickle cell anemia as a result of increased bilirubin in the bile.

Infection plays an important role in both cholelithiasis and cholecystitis. Chronic, low-grade bacterial involvement of the gallbladder produces cellular debris on which the various salts precipitate in the early stages of mixed stone formation. When mechanical obstruction of the cystic duct occurs, invasive infection of the distended gallbladder is common. Bacteria of intestinal origin (streptococci, coliform bacteria, and staphylococci) can be cultured from about half of calculous gallbladders removed at operation.

Gallstones are asymptomatic in two-thirds of cases, being discovered incidentally at operation or autopsy or on x-ray films. The management of asymptomatic gallstones is controversial, but most surgeons advise prophylactic removal of the gallbladder if the patient is a reasonably good surgical risk. This opinion is based on the fact that at least one-third to one-half of these patients subsequently develop severe symptoms or complications such as acute cholecystitis or common duct stone. The chance of developing cancer of the gallbladder in the presence of cholelithiasis is probably slightly less than 1%.

ACUTE CHOLECYSTITIS

Cholecystitis is associated with gallstones in over 90% of cases. Acute cholecystitis is usually superimposed on a chronic process and is precipitated by obstruction of the cystic duct by a stone (or, rarely, by edema in the absence of calculi). There is rapid development of a tense, edematous, inflamed gallbladder. Infection often follows as a result of invasion by resident organisms.

Clinical Findings.
A. Symptoms and Signs: A past history suggestive of chronic cholecystitis (see p. 312) can often be obtained. The acute attack is frequently precipitated by a heavy meal and begins with right upper quadrant pain which usually radiates to the right infrascapular region. Pain is agonizingly severe, prostrating, and associated with vomiting.

Right upper quadrant tenderness is invariably present, and in most cases is associated with local muscle spasm and rebound tenderness. The tensely distended gallbladder is frequently palpable. Minimal jaundice is occasionally present in the absence of common duct obstruction. Marked jaundice indicates choledocholithiasis or liver damage.

Low-grade or moderate fever is present.
B. Laboratory Findings: Moderate leukocytosis (10,000-20,000 WBC) is typical. Serum bilirubin levels of 1-4 mg./100 ml.

may be seen in the absence of common duct obstruction; clinical jaundice appears when the bilirubin exceeds 2.5 mg./100 ml. Slight elevation of the serum amylase may rarely be noted.
C. X-ray Findings: Gallstones are found on plain abdominal x-rays in about 25% of cases of acute cholecystitis. Intravenous cholecystography may be a useful emergency diagnostic procedure. If the gallbladder fills, acute cholecystitis is ruled out.

Differential Diagnosis.

The disorders most likely to be confused with acute cholecystitis are perforated peptic ulcer (see p. 284), acute pancreatitis (see p. 328), appendicitis in a high-lying appendix (see p. 275), perforated carcinoma or diverticulum of the hepatic flexure, liver abscess, liver congestion, acute viral hepatitis, and pneumonia with pleurisy on the right side. The diagnosis of uncomplicated acute cholecystitis is usually not difficult because of the definite localization of pain and tenderness in the right upper quadrant and the characteristic right infrascapular radiation.

Complications.

The following complications of acute cholecystitis are more likely to occur in diabetics and in patients over 50 with a long history of biliary tract disease. Common duct stones are also more frequent in the latter group.
A. Gangrene of the Gallbladder: Continued marked or progressive right upper quadrant pain, tenderness, muscle spasm, fever, and leukocytosis after 24-48 hours are suggestive of severe inflammation and possible gangrene of the gallbladder. Necrosis may occasionally develop without definite signs, especially in the obese abdomen.

Minor perforations of the gallbladder are frequently walled off by omentum, with or without the development of a localized abscess. Perforation into the free peritoneal cavity results in bile peritonitis which may be associated with shock and rapid deterioration of the patient's condition. Rarely, considerable sterile bile may accumulate intraperitoneally without marked signs.
B. Cholangitis: Intermittent high fever and chills are the major signs. Common duct stone may be a contributing cause.

Treatment.

Acute cholecystitis will subside on a conservative regimen in the majority of cases. Cholecystectomy can then be scheduled 6 weeks to 3 months later when the patient's general condition is optimal and the technical difficulties of operation minimized.* If, as occasionally happens, recurrent acute symptoms develop during this waiting period, cholecystectomy is indicated without further delay. When a program of conservative therapy is elected for acute cholecystitis, all patients (particularly the diabetic, the obese, and the elderly) must be watched carefully for signs of gangrene of the gallbladder.

Operation for acute cholecystitis is mandatory when there is evidence suggestive of gangrene or perforation. Operation during the acute stage is also justified as a means of reducing over-all morbidity in good risk patients in whom the diagnosis is unequivocal.

It is best to defer operation, if possible, in the presence of acute pancreatitis or common duct stone.

A. Conservative Treatment: During the acute period while the patient is being evaluated, the abdominal examination and WBC should be repeated several times daily. The principles of treatment are the same as in acute peritonitis (see p. 260), with the addition of an anticholinergic drug such as parenteral atropine or oral belladonna. Meperidine (Demerol®) is the analgesic of choice, since morphine produces spasm of the sphincter of Oddi. Antibiotics (e.g., penicillin and strepto-mycin; or tetracycline alone; or the 3 drugs together in severe cases) are administered in all except mild, rapidly subsiding cases.

B. Surgical Treatment: When surgery is elected for acute chole-cystitis, cholecystectomy is the operation of choice. The common duct should also be explored if indicated (see p. 316). In the poor risk patient or when technical difficulties with cholecystectomy arise, cholecystostomy is the safest proce-dure.

CHRONIC CHOLECYSTITIS

Clinical Findings.

A. Symptoms and Signs: When significant complaints occur they fall into 2 general categories: (1) chronic dyspepsia with belching, flatulence, nausea, and other nondescript forms of indigestion, usually aggravated by fatty foods and heavy meals; and (2) recurrent "biliary colic" characterized by attacks of right upper quadrant pain radiating to the right infrascapular region, lasting a few minutes or hours, occasionally accom-panied by vomiting, and often precipitated by dietary indis-cretion.

There are no specific physical findings except for transient, mild right upper quadrant tenderness during attacks of biliary colic. If hydrops of the gallbladder is present (rare), the tense, nontender organ can usually be palpated with ease.

B. Laboratory Findings: None are diagnostic. Serum bilirubin and liver function tests should be done, especially if common duct stone or liver disease is suspected.

C. X-ray Findings: Oral cholecystography is the most important diagnostic procedure. The presence of gallstones on plain films or cholecystography is presumptive evidence of chole-cystitis. When there is simply nonfilling of the gallbladder, cholecystography is repeated with a double dose of the test medium. Alternatively, an intravenous cholecystogram can be ordered, particularly if common duct stone is suspected. If the gallbladder fails to visualize on the second examination, it is probably diseased. Cholecystography is unreliable when there is significant liver dysfunction (BSP retention greater than 20%), common duct obstruction (serum bilirubin above 5%), malabsorption of the test material, or in the presence of an acute abdomen from any cause.

The noncalculous gallbladder which fills poorly and empties sluggishly is not a surgical problem, but because small stones

may be easily overlooked in such cases, cholecystography should be repeated if symptoms are especially suggestive of gallbladder disease. Sensitivity to iodine is the only contra-indication to cholecystography.

Differential Diagnosis.

If there are attacks of typical biliary colic and x-ray evidence of cholelithiasis or a nonfunctioning gallbladder, the diagnosis is not difficult. When nonspecific dyspeptic symptoms are the chief complaint of the patient, it is necessary to consider other gastro-intestinal conditions. Among these are nervous dyspepsia, peptic ulcer, gastritis, chronic pancreatitis, and carcinoma of the stomach, pancreas, hepatic flexure, liver, or gallbladder. It is a good rule to obtain an upper gastrointestinal barium study on patients with suspected gallbladder disease because of the frequent coexist-ence of other pathology (especially peptic ulcer).

Complications.

The complications of chronic cholecystitis with cholelithiasis include acute cholecystitis, common duct stone, cholecystenteric fistula, pancreatitis, and carcinoma of the gallbladder.

Treatment.

A. Medical Measures: Symptomatic treatment of chronic chole-cystitis is indicated when operation is contraindicated or re-fused. This consists of a low-fat diet (with avoidance of all foods discovered to cause distress), an anticholinergic agent such as tincture of belladonna (see p. 612), and a bile acid preparation such as one of the following: (1) Dehydrocholic Acid Tablets, U.S.P. (Decholin®), 0.5 Gm. (7$\frac{1}{2}$ gr.) t.i.d., p.c.; or (2) Ox Bile Extract Capsules (or Tablets), N.F., 0.3-0.6 Gm. (5-10 gr.) t.i.d., p.c.

B. Surgical Treatment: The most satisfactory treatment for symptomatic, calculous cholecystitis is removal of the gall-bladder. Choledochostomy may also be indicated (see p. 316).

Postoperative Complications of Cholecystectomy.

The incidence of postoperative complications is about 8%. Most of the complications are common to other major abdominal opera-tions. The following are peculiar to gallbladder surgery:

A. Bile Drainage: A Penrose (rubber tissue) drain is customarily placed in the subhepatic space near the foramen of Winslow and brought out through a stab wound at operation. This drain is usually shortened on the fifth and sixth days and removed on the seventh or eighth day. Persistent drainage of bile along the drain indicates interruption of an accessory bile duct, leakage of the cystic duct stump, or damage to the common duct. If the stools are at the same time acholic, common duct obstruc-tion by ligature or calculus must be suspected and early ex-ploration considered. Signs of peritoneal irritation associated with excessive bile drainage indicate bile peritonitis requiring immediate laparotomy to stop the leak or improve drainage. In the absence of peritonitis or common duct injury or obstruc-tion, a biliary fistula will tend to close and may be treated ex-pectantly.

B. Jaundice: Jaundice which develops in the immediate postoperative period suggests common duct injury, retained common duct stone, or hepatic failure. Injured ducts should usually be repaired at once. Retained stones, if small, will sometimes pass on the biliary flush routine (see p. 317). In partial obstruction, intravenous cholangiography will delineate the ducts.

C. "Postcholecystectomy Syndrome": This vague but widely used term is used to denote that situation in which the patient continues to have symptoms of chronic biliary tract disease after cholecystectomy. The cause may be common duct stone, a long remnant of cystic duct, chronic pancreatitis, or stenosis of the sphincter of Oddi. Functional or organic gastrointestinal disturbance existing preoperatively - and for which the gallbladder was mistakenly removed - should be suspected. The patient whose gallbladder was removed for noncalculous chronic cholecystitis is a likely candidate for this "syndrome." Treatment depends on etiology. If none is found, a medical program as for chronic cholecystitis can be tried (see p. 313).

Prognosis.

The over-all mortality following cholecystectomy is less than 1%. However, biliary tract surgery is more complicated and hazardous in elderly patients; in patients over 70, cholecystectomy probably has a mortality of 5-10%.

Following a properly performed operation, the patient usually is asymptomatic and requires no special diet or regimen.

CHOLEDOCHOLITHIASIS

About 10% of patients with gallstones have choledocholithiasis. The percentage rises with age, and the incidence in elderly people may be as high as 50%. Common duct stones usually originate in the gallbladder but may also form in the common duct. The stones are frequently "silent," as no symptoms result unless there is some obstruction.

Clinical Findings.

A. Symptoms and Signs: A history suggestive of chronic cholecystitis can usually be obtained. The additional features which suggest the presence of a common duct stone are (1) frequently recurring attacks of biliary colic, (2) chills and fever associated with the attacks of colic, and (3) a history of jaundice. Jaundice, which may be transient, is usually first noted within 1-2 days after an attack of colic. Occasionally there is no pain associated with the jaundice.

The presence of jaundice is strong evidence for common duct stone in a patient with a history of chronic gallbladder disease. Epigastric tenderness may occur during attacks of colic. Otherwise there are no specific abdominal signs.

B. Laboratory Findings: Liver function tests should be performed on all cases (see p. 320). Bilirubinuria and elevation of serum bilirubin are present if the common duct is obstructed. Elevation of the serum alkaline phosphatase is especially suggestive

of obstructive jaundice. Because BSP retention is increased by duct obstruction, this test does not evaluate hepatocellular function under these circumstances. Prolongation of the prothrombin time begins to occur when bile is excluded for more than a few days from the gastrointestinal tract. When marked obstructive jaundice persists for several weeks, liver damage occurs and differentiation of obstructive from hepatocellular jaundice becomes progressively more difficult.

C. X-ray Findings: In the absence of significant jaundice, intravenous cholangiography will usually visualize the common duct. When jaundice is marked, plain abdominal x-rays are studied for biliary calculi.

Differential Diagnosis.

The commonest cause of obstructive jaundice is common duct stone. Next in frequency is carcinoma of the pancreas, ampulla of Vater, or common duct. Metastatic carcinoma (usually from the gastrointestinal tract) and direct extension of gallbladder cancer are other important causes of obstructive jaundice. Hepatocellular jaundice can usually be differentiated by history, clinical findings, and liver function tests.

Complications.

A. Biliary Cirrhosis: Prolonged common duct obstruction causes severe liver damage; hepatic failure or portal hypertension may be the ultimate result in untreated cases.

B. Cholangitis: The incidence of bacteria in common duct bile is 75% when calculi are present; the organisms most frequently cultured are Escherichia coli, Aerobacter aerogenes, Streptococcus faecalis, and Proteus vulgaris. Ascending infection is frequent in common duct stone, adds to liver damage, and may rarely lead to multiple liver abscesses.

C. Hypoprothrombinemia: Patients with obstructive jaundice or liver disease may bleed excessively at operation as a result of hypoprothrombinemia. If the prothrombin deficiency is due to faulty vitamin K absorption, the following preparations are of value: (Parenteral administration is preferred to ensure complete absorption.)

1. I.V. or subcut. - Give 1 of the following·
   a. Menadione Sodium Bisulfite, U.S.P. (Hykinone®, Synkayvite®), 10 mg. (1/6 gr.) daily.
   b. Phytonadione, U.S.P. (Mephyton®), 10 mg. (1/6 gr.) daily.

2. Orally - Give 1 of the following:
   a. Menadione, U.S.P., Menaphthone, B.P. (Hykinone®, Synkayvite®), 5 mg. (1/12 gr.) b.i.d., p.c. If there is obstructive jaundice, supplementary bile salts such as ox bile extract capsules or tablets (see p. 313) must be given with menadione.
   b. Phytonadione, U.S.P. (Mephyton®), 5 mg. (1/12 gr.) b.i.d.

Treatment.

Common duct stone is treated by cholecystectomy and choledochostomy.

A. **Preoperative Care:** Emergency operation is rarely necessary; a few days devoted to careful evaluation are well spent.
   1. Liver function should be evaluated thoroughly (see p. 320).
   2. Prothrombin time should be restored to normal by parenteral administration of vitamin K preparations (see above).
   3. Glycogen and protein depletion should be combatted by a high-carbohydrate, high-protein, low-fat diet providing about 50 Calories and 2 Gm. of protein/Kg. body weight.
   4. Vitamin supplements should be given (see p. ,87).
   5. Cholangitis, if present, should be controlled with antibiotics (e.g., a tetracycline, or penicillin and streptomycin; see p. 614).

B. **Indications for Common Duct Exploration:** At every operation for cholelithiasis the advisability of exploring the common duct must be considered. Operative cholangiography via the cystic duct is a very useful procedure for demonstrating common duct stone. Any of the following evidences of common duct stone may be an indication for choledochostomy:
   1. Preoperative findings suggestive of choledocholithiasis include history or presence of obstructive jaundice; frequent attacks of biliary colic; cholangitis; history of pancreatitis; and an intravenous cholangiogram showing stone, obstruction, or dilatation of the duct.
   2. Operative findings suggestive of choledocholithiasis are palpable stones in the common duct; dilated or thick-walled common duct; gallbladder stones small enough to pass through the cystic duct; and pancreatitis.

C. **Postoperative Care:**
   1. Antibiotics - Postoperative antibiotics are not administered routinely after biliary tract surgery. Cultures of the bile are always taken at operation. If biliary tract infection was present preoperatively or is apparent at operation, penicillin and streptomycin or a tetracycline is administered postoperatively until sensitivity tests on culture specimens are available.
   2. Management of the T-tube - Following choledochostomy a simple catheter or T-tube is placed in the common duct for decompression. It must be attached securely to the skin or dressing because inadvertent removal of the tube may be disastrous. A properly placed tube should drain bile at the operating table and continuously thereafter; otherwise it is blocked or dislocated. The volume of bile drainage varies from 100-1000 ml. daily (avg., 200-400 ml.). Above-average drainage may be due to obstruction at the ampulla (usually edema), increased bile output, low resistance or siphonage effect in the drainage system, or a combination of these.
   3. Cholangiography - A cholangiogram through the T-tube should be done on about the seventh or eighth postoperative day. Under fluoroscopic control a radiopaque medium (e.g., 50% Hypaque®) is aseptically and gently injected until the duct system is outlined and the medium begins to enter the duodenum. The injection of air bubbles must be avoided since on x-ray they resemble stones in the duct system. Spot films are taken. If the cholangiogram shows no stones in the common duct and the opaque medium flows freely into the

duodenum, clamp the tube overnight and remove it by simple traction on the following day. A small amount of bile frequently leaks from the tube site for a few days. A rubber tissue drain is usually placed alongside the T-tube at operation. This drain is partially withdrawn on the fifth day and shortened daily until it is removed completely on about the seventh day.

Retained Common Duct Stone After Choledochostomy.

The reported incidence of retained stone varies from 2-25%. The frequency is decreased by good operative technic and by operative cholangiography. The residual stone is usually discovered on postoperative cholangiography. Surgical removal will eventually be necessary if th stone does not pass, but the following procedures will clear th imon duct of stone in 30-40% of cases.

A. Management V   be in Place: The stone may be fragmented o   the method of Pibram (Surg., Gynec., & Obst,   er preliminary aspiration of bile, first O,   her and then 1-2 ml. of liquid paraffi   ly into the T-tube. The tube is clampe.   pressure develops, the clamp is released. This pr   may be repeated 1 or more times daily for a number   weeks.

Five ml. of a mixture of two-thirds ethyl ether and one-third ethyl alcohol may be used instead of ether. (Walters and Wesson, Surg.,Gynec.,& Obst.85:595, 1947.)

B. Management Without T-tube. A stone remaining after removal of the T-tube can often be visualized by I.V. cholangiography. The following three-day biliary flush regimen may induce passage of the stone (Best, Ann.Surg.128:348, 1948):

1. Dehydrocholic Acid Tablets, U.S.P. (Decholin®), 0.75 Gm. (11½ gr.) q.i.d. (p.c. and h.s.) for 3 days, then 0.5 Gm. (7½ gr.) b.i.d. thereafter. Patients who tend to form recurrent stones should be kept indefinitely on this or similar medication.

2. Magnesium Sulfate, U.S.P., B.P., 1 tsp. in water each morning.

3. Two tablespoonfuls of pure cream or olive oil before the noon and evening meals and h.s.

4. Glyceryl Trinitrate Tablets, U.S.P., B.P., 0.6 mg. ($\frac{1}{100}$ gr.) placed under the tongue before each meal on the first and third days.

The above procedure may also be of value while the T-tube is still in place. Under these circumstances, the tube is kept clamped to permit intraductal pressure to build up. The tube is irrigated each day with warm olive oil after 0.6.mg. ($\frac{1}{100}$ gr.) of glyceryl trinitrate has been dissolved under the tongue. The flush regimen may be repeated several times if cholangiograms reveal a persistent stone.

DIFFERENTIAL DIAGNOSIS OF JAUNDICE

The evaluation of the jaundiced patient should be thorough and expeditious. A primary objective is to distinguish "surgical" jaundice (i.e., jaundice caused by obstruction of the extrahepatic biliary system) from jaundice due to a medical condition. Liver function tests are performed routinely (see p. 320), but must be interpreted in the light of an accurate history and physical examination. A series of x-rays of the gastrointestinal tract is usually obtained. An intravenous cholangiogram may visualize the gallbladder or ductal system if the total bilirubin is less than 5 mg./100 ml. and the BSP retention less than 40%. When the diagnosis remains obscure in spite of these studies, several courses of action are open. It may be elected to observe the progress of the patient with repeated stool examinations for bile and weekly liver function tests. A needle biopsy of the liver may be considered unless contraindicated by a bleeding tendency or the possibility that marked biliary obstruction will cause bile leakage from a liver puncture. In spite of all measures, diagnosis is impossible in about 5% of cases and laparotomy is necessary to distinguish surgically correctible jaundice from other types.

Classification of Jaundice.
A. Hepatocellular: Caused by cirrhosis, toxins (viral or chemical), abscess (amebic, pyogenic, fungal), anoxia (congestive heart failure), or tumors.
B. Obstructive: Caused by either extrahepatic obstruction or intrahepatic obstruction.
C. Hemolytic: Caused by excessive blood destruction.
D. Congenital hyperbilirubinemia (constitutional hepatic dysfunction and Dubin-Johnson syndrome).

Hepatocellular vs. Obstructive Jaundice.
It is frequently difficult to distinguish hepatocellular from extrahepatic obstructive jaundice when obstruction has been present for several weeks with resultant hepatic damage. In some cases obstructive jaundice may persist for 4-6 weeks before liver injury occurs, but in other instances a shorter interval is sufficient.
A. Clinical Findings Suggestive of Hepatocellular Jaundice:
  1. Viral hepatitis - Characteristic features of viral jaundice are vague upper abdominal pain, nausea, low-grade fever, diffuse liver tenderness and enlargement, and a history of contact with infectious hepatitis or of receiving an injection of a blood product 45-120 days previously. Pruritus is rare.
  2. Portal cirrhosis - Cirrhotics usually have a history of known chronic liver disease, and alcoholism is frequent. Chronic weakness and dyspepsia are common symptoms. Cirrhosis may be associated with palmar erythema, spider hemangiomas, gynecomastia, testicular atrophy, splenomegaly, ascites, dilated abdominal veins, hemorrhoids, and esophageal or gastric varices.
B. Clinical Findings Suggestive of Extrahepatic Obstructive Jaundice: Upper abdominal pain occurs in the majority of these patients. Since choledocholithiasis is the commonest cause of obstructive jaundice, the pain often takes the form of biliary

colic. Jaundice usually follows an attack of pain. A past history of chronic cholecystitis is frequent. Pruritus and clay-colored stools are characteristic of obstruction. Cholangitis secondary to obstruction may cause chills and fever. Fluctuating jaundice is usually caused by stone, whereas insidious, progressive jaundice is more characteristic of malignancy. A tensely distended, nontender gallbladder in the presence of jaundice signifies extrinsic (usually neoplastic) obstruction of the common duct (Courvoisier's law).

C. Laboratory Findings: See p. 320.

BENIGN COMMON DUCT STRICTURES

These are relatively rare. About 90% are caused by operative injury during cholecystectomy, and in about 30% of these patients the disorder is fatal. Jaundice or excessive bile drainage in the postoperative period after biliary tract surgery suggests the diagnosis; prompt reoperation should be considered in these cases. Delayed onset of jaundice or cholangitis after biliary tract surgery may be due to stricture but is more likely to be the result of retained or recurrent common duct stone. Inflammatory strictures of the bile duct are occasionally caused by chronic pancreatitis, postbulbar peptic ulcer, or common duct stone.

Surgical relief of the stricture should be recommended before liver damage becomes severe. Operations for repair of traumatic stricture require great technical skill. Even in the best hands, satisfactory results can be expected in little more than half of these patients. The various procedures employed are end-to-end anastomosis of the bile duct, choledochoduodenostomy, and choledocho-jejunostomy. Multiple operations are often necessary because of recurrent stricture.

PORTAL HYPERTENSION

Portal hypertension is caused by obstruction of the portal vein. In 80% of cases the block is intrahepatic and is a late result of cirrhosis. Extrahepatic obstruction by thrombosis, atresia, or cavernomatous transformation accounts for the remaining cases. Portal hypertension is of surgical significance when complicated by bleeding from esophageal or gastric varices, by intractable ascites, or by severe hypersplenism (see p. 337). The incidence of hemorrhage from esophageal varices in cirrhosis is about 30%, and the mortality in those patients who bleed ranges from 50-70%.

Clinical Findings.

The diagnosis of portal hypertension is rarely difficult. There is often a history of massive upper gastrointestinal hemorrhage. In cirrhosis, 1 or more of the following signs is present: esophageal varices, hepatomegaly, splenomegaly, thrombocytopenia, leukopenia, jaundice, or ascites. Liver function tests show impairment. The findings in extrahepatic block are similar except that jaundice, ascites, and markedly impaired liver function are rare.

Laboratory Examinations in
Hepatocellular and Obstructive Jaundice

Tests*	Hepatocellular Jaundice	Uncomplicated Obstructive Jaundice
Bilirubin		
Direct	Increased late (frequently)	Increased early
Indirect	Increased early	Increased late
Urine bilirubin	Increased	Increased
Urine urobilinogen	Increased	Markedly decreased in complete obstruction
Stool urobilin	Unchanged or lowered	Decreased
Bromsulphalein retention	Increased	Increased
Cephalin flocculation	+++ to ++++	Not over ++, usually 0 to +
Thymol turbidity	Over 4 units	Not over 4 units
Serum protein·	Albumin decreased if damage severe; A/G ratio reversed	Unchanged
Alkaline phosphatase	Increased if damage severe	Increased
Cholesterol		
Total	Decreased if damage severe	Increased
Esters	Decreased if damage severe	Increased
Prothrombin time†	Prolonged if damage severe	Prolonged if obstruction marked
Serum glutamic pyruvic transaminase (SGPT) and serum glutamic oxaloacetic transaminase (SGOT) titers‡	Increased in viral hepatitis	Usually unchanged, rarely increased

*For normal values, see back endsheets.
†If there is no severe hepatocellular damage, the prothrombin time is restored to normal in 24 hours by the I.M. injection of 5 mg. (1/12 gr.) of vitamin K.
‡If SGOT and other liver tests are equivocal, serum iron concentration should be determined. Serum iron values greater than 180 mcg./100 ml. are suggestive of acute hepatitis; values greater than 200 mcg./100 ml. are practically diagnostic of acute hepatitis when hemolytic anemia and hemochromatosis have been excluded.

The diagnostic work-up should include, in addition to routine examinations, an upper gastrointestinal series (with careful observation for esophageal or gastric varices or peptic ulcer), hepatic function tests (see p. 320), and platelet count. Liver biopsy, bone marrow study, and esophagoscopy are occasionally indicated. When extrahepatic block is seriously considered, portal venography (by splenic puncture) may demonstrate the block and confirm the diagnosis, but results must be interpreted with care for the portal vein may also fail to fill in high-grade intrahepatic obstruction.

Treatment.

A. Emergency Treatment of Bleeding Esophageal Varices: Varices are established as the source of bleeding by an immediate and thorough investigation, and general measures are instituted as for other forms of massive gastrointestinal hemorrhage (see p. 280). Additional specific therapy consists of balloon tamponade of the varices and prevention of ammonia intoxication.

1. Sengstaken tube (see p. 322) - Used for arresting hemorrhage from esophageal varices.

   a. Technic of introducing tube -

      (1) Attach the tubing of a BP manometer to a "Y" glass tube. Connect 1 limb of the "Y" to the esophageal balloon, the other to a BP bulb.

      (2) Passing tube - Lubricate the tube with jelly and pass it through the nares into the stomach to the "50 cm." mark. Facilitate swallowing with sips of water.

      (3) Securing tube in esophagus -

         (a) Inflate stomach balloon with 200 cc. of air by means of a syringe, and then clamp this tube.

         (b) Withdraw the Sengstaken tube until resistance is felt.

         (c) Inflate the esophageal balloon to a pressure of 25 mm. Hg (about 40-60 cc. of air). If 150-200 cc. of air are required to achieve a pressure of 25 mm., deflate balloons and readjust position. Esophageal contractions and respiratory and cardiac impulses may intermittently raise pressure to 70 mm. Hg, but minimal pressure of 25 mm. should be maintained. Clamp tube of esophageal balloon. Check pressure frequently to detect leakage.

   b. Maintenance of tube - Irrigate stomach with 50 ml. warm saline and then aspirate all contents. Connect stomach tube to constant suction for 24 hours. Irrigate tube hourly. If there is no evidence of bleeding after 48 hours, deflate the balloons in situ for 12 hours. If no further bleeding occurs, remove gently. Observe carefully for further bleeding.
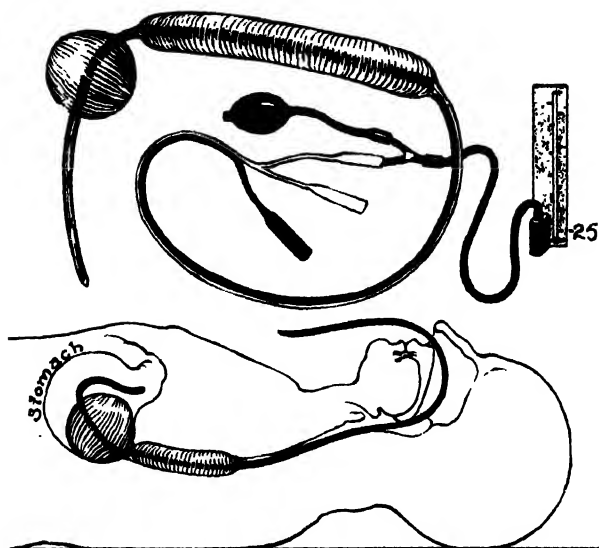
Sengstaken Tube. Reproduced, with permission, from Krupp, Sweet, Jawetz, and Armstrong: Physician's Handbook, Eleventh Edition.

2. Prevention of ammonia intoxication -
   a. Catharsis - Blood is removed promptly from the gastro-intestinal tract by administration of milk of magnesia, 60 ml. every 2-4 hours, through the Sengstaken tube, until the bowel is clear. Cleansing enemas may also be employed if necessary.
   b. Intestinal antisepsis - Through the Sengstaken tube, give neomycin, 1 Gm. every hour for 4 hours, then every 4 hours for 2 days. Neomycin, 1 Gm. every 6 hours, is continued thereafter as a maintenance dose as long as necessary to combat ammonia intoxication. Reduction of bacterial flora of the bowel by oral neomycin minimizes the production and absorption of ammonia.
3. Treatment of ammonia intoxication - See p. 324.
4. Indications for operation in bleeding varices - If bleeding cannot be controlled by the Sengstaken tube and the patient is a reasonable operative risk (see below), an emergency portacaval or splenorenal shunt should be performed. An alternative approach is the transesophageal ligation of varices followed in a few weeks by an elective portacaval shunt. The mortality in emergency operations for bleeding varices is high (25-50%), but this is acceptable in view of the seriousness of the disorder.

B. Portacaval Shunt: A history of bleeding from varices is an indication for elective portacaval shunt if liver function tests show acceptable hepatic reserve, i.e., if the serum albumin level is above 3.5 Gm./100 ml.; BSP retention is less than 30% one-half hour after injection; prothrombin time is not in excess of 4 seconds above the normal control (Link and Shapiro method, undiluted); cephalin flocculation is not more than 2+; and serum bilirubin is under 1.5 mg./100 ml. (criteria of Blakemore). In posthepatitis cirrhosis, the cephalin flocculation, BSP retention, and bilirubin levels may be somewhat more abnormal without affecting the operative risk adversely.

Chronic ascites is also an indication for portacaval shunt (side-to-side) in selected cases.

1. Preoperative care - A period of several weeks (or even months) of intensive care will frequently result in sufficient improvement to permit surgical intervention. A low-sodium, 3000 Calorie diet containing 150 Gm. of protein and 75-100 Gm. of fat is prescribed. Commercially available protein or amino acid supplements may be used, but protein intake should be reduced to 0.5 Gm./Kg. body weight if ammonia intoxication is present. Therapeutic doses of vitamin B complex, vitamin C (see p. 87), and vitamin K (see p. 315) are administered. Anemia is corrected by transfusion of whole blood or packed red cells Ascites, if present, should be stabilized or decreasing before operation is undertaken.

2. Shunt operations - The only successful procedures for the relief of portal hypertension are side-to-side or end-to-side anastomosis of the portal vein to the vena cava, or end-to-side anastomosis of the splenic vein to the left renal vein after splenectomy. In extrahepatic block, the splenorenal type of shunt is necessary. It is possible that the side-to-side portacaval shunt will prove superior to the end-to-side, especially in ascites, but this matter is as yet unsettled.

3. Postoperative care - Liver failure is the most serious complication; it occurs postoperatively in 15-20% of patients with cirrhosis, and is fatal in about one-third of those affected. It can be minimized by the following postoperative regimen: (1) Oxygen by nasal catheter at 6-7 L./min. for 24-48 hours; and (2) a minimum of 250 Gm. of glucose daily by vein (as 10 or 25% solution) until oral intake of the preoperative diet (see above) is tolerated. Preoperative vitamin therapy is continued.

If liver failure develops, oral protein intake is stopped or limited to 0.5 Gm./Kg. body weight per day; glucose intake (usually given intravenously) is increased to 300 or more Gm. daily; oral neomycin is administered; and intravenous arginine is used as indicated (see p. 325). Transfusions are given as needed to maintain Hgb. above 12 Gm.%.

Some patients with portacaval shunt tend to develop recurrent ammonia intoxication after heavy protein meals. This may be controlled by a low-protein diet (40 Gm./day) and intermittent administration of neomycin, 0.5 Gm. orally q.i.d. every 3 days for an indefinite period.

Prognosis.

Shunt operations have a mortality of 7-15%. Splenorenal shunt for extrahepatic block is associated with the lowest mortality. About 80% of patients will show reduction in the size of the varices, and liver function may improve after a satisfactory shunt. Recurrent (usually controllable) bleeding occurs in 15-20% of cases; this is more frequent after splenorenal than after portacaval shunts.

AMMONIA INTOXICATION AND HEPATIC COMA

The syndrome of liver failure occurs most often in patients with chronic hepatic disease who have had gastrointestinal hemorrhage or a portacaval shunt. It may also develop in cirrhotics after major operation or as a terminal stage. Symptoms and signs are largely caused by ammonia intoxication, and the diagnosis is confirmed by the finding of elevated ammonia levels in blood or spinal fluid. The major source of ammonia is the gastrointestinal tract, where the ion is produced by the action of the intestinal bacteria on nitrogenous material (especially blood and protein). Normally, ammonia is detoxified by conversion to urea in the liver and, to a lesser extent, by transamination reactions in the peripheral tissues. A damaged liver cannot cope with an increased ammonia load such as results in bleeding esophageal varices. After portacaval shunt and in patients with portal hypertension who have developed collateral portal-systemic channels, the portal blood by-passes the liver and carries ammonia directly into the general circulation. These patients may have episodic ammonia intoxication related to heavy meals.

Clinical Findings.

There is usually an obvious precipitating cause such as massive gastrointestinal hemorrhage, severe surgical stress in a cirrhotic, or excessive protein intake in a patient with a portacaval shunt. The administration of ammonium salts (especially ammonium chloride) to a patient with depressed liver function may also produce ammonia intoxication.

There are various states of ammonia intoxication, ranging from mental confusion, dysarthria, and irrational behavior to lethargy, somnolence, stupor, and frank coma. One of the early characteristic signs is a flapping tremor of the hands which also occurs in uremia and respiratory acidosis. In the early stages of liver failure the deep tendon reflexes are hyperactive. There are also increased muscle tone, restlessness, and twitching movements. In the late phases, generalized or focal convulsions may accompany the stupor. As coma deepens the respirations become stertorous and the pupils dilated; the muscles become flaccid prior to death. Progressive jaundice and increasing ascites are often noted. Blood and spinal fluid ammonia levels are elevated.

Treatment.

If ammonia intoxication is threatened or precipitated by massive bleeding, the measures outlined for the treatment of tamponade and varices are instituted (see p. 321).

Under other circumstances, the general principles of management include:

A. Limitation of Protein Intake: Oral protein is stopped altogether or limited to 0.5 Gm./Kg. body weight daily.

B. High Carbohydrate Intake: Carbohydrate intake is maintained at 300 Gm. daily, usually by administration of supplementary glucose (10-25% solution) I.V.

C. Intestinal Antisepsis: Neomycin, 1 Gm. every 6 hours orally or by tube, until ammonia levels are normal. Neomycin, 1% solution, can be given by retention enema (500-1000 ml.) if oral administration is impossible.

D. Arginine Therapy: Intravenous arginine reduces the ammonia level in the blood and may be used as one of the initial measures in severe ammonia intoxication. Its chief value is to tide the patient over until the sources of ammonia production in the gastrointestinal tract can be eliminated by catharsis or neomycin. If arginine is to be effective, it should be noted within 48 hours or so. Give 500 ml. of 5% arginine hydrochloride in 10% glucose solution I.V. over a two-hour period. Repeat every 8-12 hours as necessary.

Prognosis.

The mortality in hepatic failure is at least 30%. This can be reduced only by early and intensive treatment.

TUMORS OF THE LIVER AND BILIARY TRACT

Benign tumors of the liver and biliary tract are rare and are usually incidental findings at operation or autopsy. The commonest benign liver tumor is cavernous hemangioma, which usually presents beneath the liver capsule as multiple purplish, nodular, compressible masses. No treatment is necessary. Papillomas, adenomas, and lipomas occur throughout the biliary system and are treated by excision. They may be visualized by cholecystography.

Primary Carcinoma of the Liver.

This is rare in the United States but relatively common in Africa and the Orient. It is primarily a disease of adults 40-50 years old, but a peak incidence also occurs in infancy and childhood. It is more frequent in males.

The possible etiologic factors are cirrhosis, malnutrition, parasites, chronic irritation, hemochromatosis, and congenital defects. Three histologic types can be distinguished: malignant hepatoma, malignant cholangioma, and a mixed type (malignant cholangiohepatoma). Hepatoma is usually the most rapidly progressive.

Early in the disease there are no pathognomonic symptoms. Later, abdominal pain and enlargement of the liver are noted. Occasionally there is fever. A needle or open liver biopsy establishes the diagnosis.

The prognosis is grave but not always hopeless, as it is possible in rare cases to resect the lesion. Metastatic liver cancer is very common and must be differentiated from primary tumor.

Adenocarcinoma of the Gallbladder.

About two-thirds of carcinomas of the extrahepatic biliary system arise in the gallbladder. The majority of patients are over 50, and 75% are women. Gallstones are present in 90% of cases and are considered the most important etiologic factor. This is one of the reasons why prophylactic removal of the calculous gallbladder is advisable unless there is some contraindication (see p. 309).

The early symptoms are those of chronic cholecystitis, which is invariably present (see p. 312). Later, pain is often continuous, and a mass in the gallbladder region may be palpable. Jaundice usually occurs eventually.

Cancer of the gallbladder is almost always fatal, although a few five-year survivals have been reported. Radical cholecystectomy with resection of the gallbladder bed or partial hepatectomy should be attempted when the tumor is sufficiently localized.

PYOGENIC LIVER ABSCESS

Pyogenic abscess of the liver is usually secondary to intraperitoneal sepsis. The intestinal flora is commonly involved. Malaise, fever, and right or left upper quadrant pain are usually present. The pain often radiates to the top of the ipsilateral shoulder. Prostration and high spiking fever occur as the infection progresses. Although local signs depend somewhat upon the location of the abscess, an enlarged, tender liver and direct shock tenderness over the lower rib cage are frequently found. Leukocytosis is marked and may aid in differentiation from uncomplicated amebic abscess, in which the WBC usually shows minimal elevation. Jaundice is a late sign. Upright chest and abdominal films should be scrutinized for such suggestive signs as elevation of the diaphragm, fluid in the chest, enlargement or tumefaction of the liver, and abscess cavity with air fluid level.

Treatment is by incision and drainage with care to avoid contamination of the pleural and peritoneal cavities. Needle aspiration should be done only under direct vision at exploration. Antimicrobial therapy is based on culture and sensitivity studies (see p. 310).

AMEBIC LIVER ABSCESS

Clinical Findings.

Amebic abscess is caused by trophozoites of Entamoeba histolytica which reach the liver from the cecal region by way of the portal system. It occurs in less than 3% of persons with amebiasis, and is most common in young adult males. The most frequent location is in the right lobe near the dome. Abscess formation is preceded by amebic hepatitis. The symptoms and signs of amebic abscess are similar to those of pyogenic abscess. A history of dysentery is obtained and amebas in the stools are demonstrated in only 20-25% of cases. The WBC is slightly elevated and liver function tests are usually normal unless the abscess becomes chronic, in which case increasing leukocytosis and hepatic dysfunction become apparent. The complement fixation test for amebiasis is useful.

The complications of hepatic amebiasis are (1) secondary infection of the abscess, (2) pleuropulmonary complications (empyema, bronchohepatic fistula, pulmonary abscess), and (3) rupture into the peritoneal cavity. Exploratory needle puncture of the liver is employed when the diagnosis is doubtful or when secondary infection of the abscess is suspected.

Treatment.
A. Medical Measures: The following therapeutic routine is applicable in the majority of cases. If a clinical diagnosis of amebic abscess is made and there is no indication for immediate surgery, the patient is given hydroxychloroquine sulfate (Plaquenil®), 0.9 Gm. (14 gr.) daily for 10 days. If secondary infection is suspected, the patient should also receive penicillin and streptomycin or a tetracycline. If there is improvement in these 10 days, 0.6 Gm. (10 gr.) of hydroxychloroquine is administered daily for 20 more days. The patient may then be considered "cured," but he must be watched for recurrence.

If there is no improvement in the first 10 days of chloroquine therapy, an exploratory puncture of the liver under local anesthesia is done with a spinal needle, using strict aseptic technic. Care must be taken to avoid the pleural and peritoneal cavities. If amebic pus (usually reddish-brown) is obtained, the abscess is evacuated completely with a small trocar (14-16 gauge). The pus is cultured and smeared for pyogenic bacteria and amebas. No antiamebic drug is injected into the cavity. Systemic drug therapy is continued. Repeat aspiration may be required. Intestinal amebiasis should be assumed to be present, and treated with one of the iodine-containing drugs (Vioform®, Chiniofon®, or Diodoquin®) or an arsenic compound (carbarsone).

Patients who do not respond to hydroxychloroquine sulfate should be given Emetine Hydrochloride Injection, U.S.P., B.P., 65 mg. (1 gr.) daily I.M. or subcut. for 6 days. Repeat the course after 1-2 weeks if the patient responds. Emetine is contraindicated in cardiac and renal disease and in pregnancy. Use cautiously in children. During emetine therapy, confine to bed and follow pulse, BP, and Ecg. Discontinue emetine on development of toxic symptoms such as nausea, vomiting, weakness, prostration, neuritis, and myocarditis.
B. Surgical Treatment: Surgical drainage, by an extrapleural, extraperitoneal route, is employed only when 1 of the complications of amebic abscess occurs or when the abscess cannot be satisfactorily evacuated by trocar.

DISEASES OF THE PANCREAS

ANNULAR PANCREAS

The nature and severity of the symptoms produced by this congenital lesion are related to the degree of duodenal obstruction which it causes. When obstruction is marked, persistent vomiting

occurs immediately after birth. Mild duodenal narrowing may not cause symptoms until adulthood. The 4 major complications accompanying the anomaly are duodenal obstruction, peptic ulcer, acute or chronic pancreatitis, and biliary obstruction. Frank peptic ulceration in the duodenum or stomach accompanied by typical ulcer symptoms is present in the majority of adult patients. The diagnosis is confirmed by barium studies.

Treatment is surgical. In infants and in some adults, duodenojejunostomy is performed. Partial gastrectomy is advisable if peptic ulcer is present. It is important to avoid a direct attack on the pancreas itself; most of the surgical fatalities have occurred as a result of attempts to divide the pancreatic ring.

ACUTE PANCREATITIS

The cause of acute pancreatitis is not clearly established and may not be the same in all cases. It is probably precipitated by increased intraductal pressure such as occurs (1) in pancreatic duct obstruction, (2) when pancreatic secretion is copious after a heavy meal, or (3) when bile flows back into the pancreatic ductal system through a common channel at the junction of the common and pancreatic ducts. Pressure sufficient to rupture the tiny ductules in the pancreas releases pancreatic enzymes into the tissues, where they are activated and produce a severe inflammatory reaction. Two varieties occur: acute edematous pancreatitis and the more severe, fulminating, and often fatal acute hemorrhagic pancreatitis.

Clinical Findings.

Although this disorder may occur in any age group, it is most frequently seen in persons between 30 and 50. A past history of chronic cholecystitis or previous attacks of acute pancreatitis can often be obtained. Many patients with acute pancreatitis are alcoholics. Postoperative pancreatitis, particularly after gastroduodenal or biliary tract surgery, is not unusual.

A. Symptoms and Signs: In acute edematous pancreatitis the onset is relatively rapid, with severe epigastric pain which is usually referred through to the back and lower scapular regions. Nausea and vomiting almost always occur. Abdominal signs include tenderness over the pancreas, mild muscle spasm, and diminished peristalsis. Tachycardia and low-grade fever are usually present. There may be mild, transient jaundice.

In acute hemorrhagic pancreatitis, the symptoms and signs are greatly intensified. Shock occurs and may be rapidly fatal. Edema and discoloration appear in the flanks in some cases. Hypocalcemic tetany and hyperglycemia are not unusual. If the patient survives the acute necrosis, he may form a pancreatic pseudocyst or abscess with sequestration of the pancreas.

B. Laboratory Findings: The following are suggestive of acute pancreatitis: moderate to marked leukocytosis, increased Hgb. and Hct. values indicative of hemoconcentration, increased blood sugar, and decreased serum calcium.

The elevated serum amylase level is a more specific sign of pancreatitis than any other laboratory test, but it must be

considered only a useful adjunct to clinical evaluation. It usually reaches its highest value within 24 hours of onset of acute pancreatitis, and may return to normal within 48 hours or remain elevated for several days. The degree of elevation does not correlate well with the severity of the condition. Many acute abdominal conditions and even morphine administration will also cause the serum amylase to rise (occasionally to high levels) in the absence of pancreatic disease. Serum lipase tends to rise in acute pancreatitis, reaches its greatest concentration later than the amylase, and persists longer. Urinary diastase output usually increases when serum amylase rises, and should be determined on a timed two-hour urine specimen in order to estimate the hourly quantitative excretion. Urinary diastase may be increased in the presence of normal serum amylase if renal excretion of the enzyme is efficient. When kidney function is poor, urinary diastase may fail to rise.

C. X-ray Findings: Survey films of the chest and abdomen should be obtained. Basal pneumonitis, pleurisy, or pleural effusion, usually on the left side, may accompany acute pancreatitis. The abdominal x-ray often discloses an isolated, gas-filled "sentinel loop" of small intestine in the middle or upper abdomen; or a more diffuse paralytic type of ileus may be present. Radiopaque gallstones or pancreatic calcifications may be revealed.

Differential Diagnosis.

Acute cholecystitis (see p. 310), choledocholithiasis (see p. 314), and perforated peptic ulcer (see p. 284) are most likely to be confused with pancreatitis. In each of these disorders the serum amylase may rise, but usually to a lesser extent than in acute pancreatitis. Mesenteric thrombosis (see p. 273) and acute hemorrhagic pancreatitis often produce the same type of profound collapse. The diagnosis of pancreatitis can usually be made without resort to laparotomy.

Complications.

Pancreatic pseudocyst (see p. 322) is an occasional complication of acute pancreatitis. The presence of a pseudocyst is indicated by the appearance of a mass in the region of the pancreas and, in some cases, by persistent elevation of serum amylase, WBC, and temperature. Pancreatic abscess or hemorrhage secondary to necrosis may be serious sequelae of acute hemorrhagic pancreatitis.

Treatment.

A. Medical Measures: The initial treatment of acute pancreatitis is nonoperative, and the same regimen is employed as in acute peritonitis (see p. 260). An anticholinergic drug, such as atropine, is given every 4-6 hours parenterally (see p. 612). Penicillin and streptomycin or a parenteral tetracycline may be advisable in severe cases. Hypocalcemic tetany is treated with calcium gluconate, 1 Gm. in 10% solution I.V., as often as necessary to control the manifestations.

Of great importance in acute hemorrhagic pancreatitis is the intensive treatment of the profound shock which often occurs. Loss of blood, plasma, and fluid into the retroperitoneal

space is severe, requiring multiple whole blood and plasma transfusions. Cortisone (e.g., Hydro-Cortef®), 100-200 mg. I.V. every 4-6 hours for several doses, may be of some value if BP cannot be maintained by fluid replacement and vasopressor drugs (see p. 623).

Blood and urine sugar levels should be determined and insulin administered on the basis of urine test if diabetes develops (see p. 45).

B. Surgical Treatment: Operative treatment in the acute stage is reserved for a few carefully selected patients with hemorrhagic pancreatitis who are going progressively downhill in spite of completely adequate supportive care and in whom there is the chance that drainage of fluid or necrotic debris from the lesser omental sac or the peripancreatic tissues may change the course of the disease. Such procedures may rarely avert a fatal outcome.

When acute pancreatitis is unexpectedly found on exploration for an acute abdomen, it is usually wisest to close without intervention of any kind. If the pancreatitis appears mild and cholelithiasis is present, cholecystostomy or cholecystectomy may occasionally be justified. In general, patients who receive the least intra-abdominal manipulation have the least morbidity and mortality after laparotomy for unsuspected pancreatitis.

The development of a pancreatic abscess is an indication for prompt drainage, usually through the flank. If a pseudocyst develops, it often requires surgical treatment (see p. 332).

Prognosis.

The mortality in acute hemorrhagic pancreatitis is probably about 33%, and in edematous pancreatitis about 5%, although reported statistics show a very wide range. Postoperative pancreatitis has a mortality of 40-50%. The efficacy of careful nonoperative management has been proved in a series of 82 patients treated medically with an over-all mortality of only 1.3% during the acute stage of the disease (H.L. Bockus, Gastroenterol. 34:467, 1958). All patients with pancreatitis should eventually have cholecystography, cholangiography, and upper gastrointestinal x-rays. Surgical disease of the biliary system should be corrected, and stenosis of the sphincter of Oddi, if present, should be relieved by sphincterotomy in order to reduce the likelihood of future attacks.

CHRONIC RELAPSING PANCREATITIS

'Some individuals, about a third of whom are alcoholics, have repeated attacks of pancreatitis. Progressive fibrosis and varying degrees of destruction of functioning glandular tissue occur as a result. Pancreaticolithiasis and obstruction of the duodenal end of the pancreatic duct are often present. Cholecystitis is present in about 50% of patients with chronic pancreatitis. Males are affected 6 times as often as females.

Clinical Findings.

Recurrent attacks of epigastric and left upper quadrant pain with referral to the upper left lumbar region are typical. Anorexia, nausea, vomiting, constipation, and flatulence are common. Ab-

dominal signs during attacks consist chiefly of tenderness over the pancreas, mild muscle guarding, and paralytic ileus. Serum amylase and bilirubin are often elevated during acute attacks. Glycosuria may be present. Plain films often show pancreaticolithiasis and mild ileus. A cholecystogram may reveal gallbladder disease, and upper gastrointestinal series may demonstrate a widened duodenal loop. Attacks may last only a few hours or as long as 2 weeks; pain may eventually be almost continuous.

Complications.

Narcotic addiction is common. Other frequent complications include diabetes mellitus, pancreatic pseudocyst or abscess, obstructive jaundice, steatorrhea, malnutrition, and peptic ulcer.

Treatment.

Correctible coexistent biliary tract disease should be treated surgically.

A. Medical Measures: A bland, low-fat diet (see p. 97) and anticholinergic drugs (see p. 612) should be prescribed. Alcohol is forbidden because it frequently precipitates attacks. Mild sedatives may be helpful (see p. 607). Narcotics are avoided. Malabsorption is treated with Pancreatin, N.F. (e.g., Viokase®), 2 Gm. t.i.d., p.c., orally. Every effort is made to manage the disease medically.

B. Surgical Treatment: When conservative measures fail, surgical intervention must be considered. There is no agreement as to the procedure of choice, and operations must be strictly individualized. Sphincterotomy of the sphincter of Oddi has been favored by some. When obstruction of the duodenal end of the duct can be demonstrated by operative pancreatography, resection of the tail of the pancreas with pancreaticojejunostomy may be successful. In advanced cases it may be necessary, as a last resort, to do pancreatic resection. Many other operations have been advised, but no surgical procedure is sufficiently successful to be generally recommended.

Prognosis.

This is a serious disease and often leads to chronic invalidism. The prognosis is best when patients with acute pancreatitis are carefully investigated with their first attack and are found to have some remediable condition such as chronic cholecystitis and cholelithiasis, choledocholithiasis, or stenosis of the sphincter of Oddi. Surgical relief of these aggravating conditions may prevent recurrent pancreatic disease.   •

PANCREATIC CYST

The commonest type of pancreatic cyst is the pseudocyst which is secondary to pancreatitis or trauma. It is formed by the accumulation of pancreatic juice in the retroperitoneal space or lesser sac as a result of rupture of the pancreatic ducts.

Other types of pancreatic cyst are quite rare and include (1) true cysts which are lined by cuboidal or low columnar epithelium

and contain yellow fluid; and (2) neoplastic cysts which may be benign (cystadenoma) or malignant (cystadenocarcinoma).

Clinical Findings in Pancreatic Pseudocyst.

There is a history of trauma or pancreatitis. Epigastric pain and a palpable, tense, tender mass are usually observed. Anorexia and weight loss are common. Chills and fever may occur. Jaundice is present in 5-10% of patients. Serum amylase will usually not be elevated except during an attack of pancreatitis. Diabetes is occasionally found. Plain abdominal films will show a mass, and calcium in its wall may be visible. Barium study of the stomach and colon may show compression of these organs by the cyst. The duodenal loop may be widened by a cyst of the head of the pancreas. Tumor of the kidney must in some cases be ruled out by excretory or retrograde urography.

Treatment.

Pseudocysts are rarely suitable for total removal. The measures most widely recommended are simple drainage with a Pezzer catheter (especially for poor risk patients and infected cysts); and internal drainage by cystojejunostomy using a Roux-Y limb of jejunum.

TRAUMATIC INJURIES OF THE PANCREAS

Injury to the pancreas is rare. In penetrating wounds, other structures are invariably damaged also. Operative injury to the pancreas may occur, especially during gastrectomy or splenectomy. Hemorrhage and leakage of pancreatic secretions are the commonest complications of pancreatic trauma which require treatment.

When pancreatic leakage is suspected, drainage is mandatory; the fistula which often develops as a consequence of drainage tends to close spontaneously in a few weeks or months. Meticulous attention to nutrition and fluid balance is necessary to prevent serious depletion in these patients. The pancreatic juice, if copious, should be collected in a suction reservoir and administered orally.

Blunt trauma may be followed by the delayed appearance of a pseudocyst (see above). Elevation of serum amylase after abdominal trauma is suggestive but not pathognomonic of pancreatic damage.

CARCINOMA OF THE HEAD OF THE PANCREAS AND THE PERIAMPULLARY AREA

Carcinoma is the commonest neoplasm of the pancreas. About 75% are in the head and 25% in the body and tail of the organ. Carcinomas involving the head of the pancreas, the ampulla of Vater, the common bile duct, and the duodenum are considered together because they are usually indistinguishable clinically.

Clinical Findings.

Abdominal pain, jaundice, weight loss, and a palpable gall-bladder are the most frequent findings in these tumors. Pain,

which is present in over 70%, is often vague and diffuse in the epigastrium and is rarely comparable to biliary colic. Later, more persistent, severe pain develops and often radiates to the back. This usually indicates that the lesion has spread beyond the pancreas and is inoperable. The jaundice is obstructive and must be differentiated from the hepatocellular type (see p. 320). Unfortunately, it is rarely possible to make the diagnosis before jaundice occurs. Diarrhea is seen occasionally, and thrombophlebitis in the legs is a rare sign. It is a useful clinical rule (Courvoisier's law) that jaundice associated with a palpable gallbladder is indicative of obstruction by neoplasm. On rare occasions the gallbladder may not be palpable because of cystic duct obstruction or contraction of the gallbladder secondary to chronic infection. Occult blood in the stools occurs more frequently in tumors of the ampulla. Barium study of the duodenum may show deformity such as widening or indentation of the loop.

Treatment.

Abdominal exploration is usually necessary to confirm the diagnosis and determine resectability, which is about 30%. Radical pancreaticoduodenal resection is indicated for lesions which are strictly limited to the head of the pancreas, periampullary zone, and duodenum. When radical resection is not feasible, cholecysto-jejunostomy is performed to relieve the jaundice. A gastrojejunostomy is also done if duodenal obstruction is expected to develop later.

Prognosis.

Carcinoma of the head of the pancreas has a very poor prognosis; fewer than 10% of resected cases survive 5 years. Lesions of the ampulla, common duct, and duodenum are more favorable with a five-year survival rate from 20-40% after resection. The operative mortality of radical pancreaticoduodenectomy is 10-15%.

CARCINOMA OF THE BODY AND TAIL OF THE PANCREAS

About 25% of pancreatic cancer arises in the body or tail. There are no characteristic findings in the early stages. The initial symptoms are vague epigastric or left upper quadrant distress. Anorexia and weight loss usually occur. Later, pain becomes more severe and frequently radiates through to the left lumbar region. A mass in the mid or left epigastrium may be palpable. The spontaneous development of thrombophlebitis in the legs is suggestive. The diagnosis is usually made only by abdominal exploration. Resection is rarely feasible, and cure is rarer still.

ISLET CELL TUMORS

Islet cell tumors of the pancreas may be benign (90%) or malignant, and functioning or nonfunctioning. About 75% occur in the body and tail and 25% in the head of the pancreas. Most of these neoplasms are only 1-2 cm. in diameter; multiple tumors occur in

5-10% of benign cases. Over 90% of the tumors removed at operation are functioning. This is to be expected because the diagnosis is usually suggested by hypoglycemia. The majority of patients are under 30; persons past 60 are seldom affected. Rarely, the tumors arise in aberrant pancreatic tissue in the duodenum, stomach, or gastrosplenic omentum, so that a thorough abdominal search must be made if a suspected lesion is not found in the pancreas.

Clinical Findings.
   A. Functioning Islet Cell Tumors: The symptoms produced by functioning islet cell tumors depend entirely upon excessive insulin secretion and the resultant hypoglycemia. Attacks of hypoglycemia are usually precipitated by fasting or overexertion, and often begin with a feeling of intense hunger. Agitation, perspiration, pallor, dizziness, and weakness are early symptoms which may progress to confusion, stupor, excitement, disorientation, drunken behavior, delirium, mania, tonic and clonic spasms, convulsions, and coma. Blood sugar must be below 50 mg./100 ml. during an attack to be diagnostic.
   B. Ulcerogenic Islet Cell Tumors (Zollinger-Ellison Syndrome): In 1955 Zollinger and Ellison described the syndrome of atypical ulceration frequently involving the jejunum and associated with hypersecretion of the stomach. A tendency to recurrent ulcer despite gastric surgery was also noted. They related these changes to the presence of adenoma or malignant tumor of the islet cells of the pancreas. The condition is rare, and the mechanism by which the islet cell tumors influence gastrointestinal function is not known. It has been suggested that the neoplasms in the pancreas may be only a manifestation of a more generalized endocrinopathy which is also responsible for the abnormal gastric secretion. In any event, atypical or recurrent peptic ulceration should alert the surgeon to the possibility of islet cell tumors, which are frequently multiple and malignant.

Treatment.
   Surgical exploration should be carried out when the diagnosis is established, as delay may permit mental deterioration as a result of repeated attacks of hypoglycemia. An intravenous drip of 10% glucose is started before operation and continued throughout the procedure to prevent hypoglycemia. Operative mortality is about 4% and complete relief is obtained in 90% of patients with benign tumors.

DISEASES OF THE SPLEEN

The normal functions of the spleen include the following: (1) hematopoiesis in the embryo; (2) lymphocyte formation throughout life; (3) removal of worn-out and damaged blood cells from the circulation, (4) production of antibodies, (5) humoral regulation of the

bone marrow, and (6) storage of red cells and iron. In the absence of the spleen, these functions are assumed by the remainder of the reticuloendothelial system; splenectomy has no permanent adverse effect except in those rare cases when the spleen has become an important site of extramedullary hematopoiesis (e.g., in myeloid metaplasia with myelofibrosis).

Following splenectomy there is usually a transient rise in the erythrocyte, leukocyte, and platelet counts. Postoperatively, the platelet count should be determined daily for 1 week or until it is consistently below 1 million/cu. mm. If the thrombocytes exceed 1.5 million, a systemic anticoagulant (heparin or coumarin; see p. 608) may be required to prevent spontaneous thromboses. It is not unusual for the postsplenectomy patient to be febrile for 7-10 days even though no complication is present.

Indications for Splenectomy.

The medical indications for splenectomy are frequently dependent on response to other forms of treatment. Each case must be carefully studied from the hematologic point of view. Close cooperation of the internist and surgeon is essential to the proper selection and management of these patients.

  A. Surgical Indications: Rupture of the spleen (see p. 263), splenic cysts and tumors, wandering spleen, and splenorenal shunt.

  B. Medical Indications: Primary hypersplenism (congenital hemolytic anemia, idiopathic thrombocytopenic purpura, primary splenic neutropenia, primary splenic panhematopenia), secondary hypersplenism, or acquired hemolytic anemia.

  C. Miscellaneous Indications for Splenectomy: In the occasional case of hypoplastic anemia in which the bone marrow is hyperactive and the spleen at least slightly enlarged, splenectomy often leads to improvement and occasionally to a hematologic cure. Removal of massive spleens associated with medical conditions such as Gaucher's disease, malaria, and schistosomiasis may be indicated for mechanical reasons and because mild hypersplenism is often present.

Contraindications to Splenectomy.

The current tendency is to liberalize the indications for splenectomy; this operation has proved beneficial in carefully selected cases in many disorders which were formerly thought to be contraindications. Before splenectomy is undertaken for any of the medical diseases, it must be demonstrated that the spleen is affecting the hematologic picture adversely or that mechanical symptoms justify the operation. Splenectomy should not be performed in Mediterranean anemia (thalassemia) and sickle cell anemia unless a hemolytic factor is prominent.

In general, removal of the spleen is contraindicated in pernicious anemia, polycythemia, the lymphomas, and agnogenic myeloid metaplasia.

CYSTS AND TUMORS OF THE SPLEEN

Cysts of the spleen are very rare and are of 3 types: (1) primary (true) cysts, (2) secondary cysts (pseudocysts), and (3) parasitic (echinococcus) cysts. They are usually asymptomatic unless they become large enough to cause abdominal discomfort. Calcification in the cyst wall may aid in diagnosis by x-ray. Treatment is by splenectomy.

Primary neoplasms of the spleen are even more rare than cysts. Hemangioma is the most frequent benign tumor. Sarcoma of the lymphoma group is the commonest malignancy. Splenectomy is curative in hemangioma, but five-year survival after removal of the spleen for sarcoma is unusual.

WANDERING SPLEEN

In this disorder the spleen has a long pedicle and floats free in the peritoneal cavity. It may become twisted on its pedicle. Treatment consists of splenectomy.

PRIMARY HYPERSPLENISM

Primary hypersplenism is characterized by depression (or excessive destruction) of 1 or more of the formed elements of the blood as a result of splenic overactivity. Depending on which blood elements are involved, the following conditions may result: (1) Congenital (primary) hemolytic anemia (erythrocytes), (2) idiopathic thrombocytopenic purpura (platelets), (3) primary splenic neutropenia (neutrophils), and (4) primary splenic panhematopenia (all elements). In all of these conditions except idiopathic thrombocytopenic purpura, splenectomy is the treatment of choice.

Congenital (Primary) Hemolytic Anemia (Congenital Hemolytic Jaundice; Spherocytic Jaundice).

Onset of symptoms may occur any time between infancy and 40-50 years of age. A familial history is important. The most typical feature is acholuric jaundice. Weakness and malaise are the commonest complaints except during hemolytic crises, when marked hemolysis is associated with nausea, vomiting, abdominal pain, and rapid increase in jaundice. Remissions and exacerbations are characteristic of this disease. Calcium bilirubinate biliary calculi are common as a result of high biliary bilirubin output, and may produce symptoms of cholelithiasis. Splenomegaly is usually present, but the spleen may not be palpable in mild cases.

The clinical diagnosis requires laboratory confirmation. Microcytic anemia is present, and erythrocyte counts below 1 million may be seen after hemolytic crises. Spherocytosis, reticulocytosis of 5-20%, and increased red cell fragility are found. Bone marrow studies showing erythroid hyperplasia are essential to rule out other forms of anemia. Indirect serum bilirubin and urine and stool urobilin are increased. Bile is not present in the urine.

Splenectomy is the only satisfactory treatment. Cortisone or corticotropin may be useful to tide a patient over a hemolytic crisis.

Preoperative transfusions are contraindicated because of the danger of precipitating a hemolytic crisis. As soon as the splenic artery is interrupted at operation, blood replacement can be safely started. The results of splenectomy are immediate and dramatic.

Idiopathic Thrombocytopenic Purpura.

The cause of idiopathic thrombocytopenic purpura is an autoimmune process in most if not all cases. Platelet agglutinins are commonly found. This disease is seen in 2 forms: (1) an acute, self-limiting illness in infants and children in which remissions usually occur after a few weeks or in some cases as long as a year; and (2) a chronic process in young adults, more commonly women, which may have a remission but often requires splenectomy.

Symptoms and signs vary with the severity of the thrombocytopenia and consist of easy bruising, an otherwise asymptomatic skin rash (petechiae), bleeding from the body orifices or into the subcutaneous tissues, weakness, and loss of weight. Hemorrhage into the brain is the most serious complication, and occurs in about 10% of cases. The spleen is rarely enlarged; if splenomegaly is present, some other disease such as leukemia should be suspected.

Thrombocytopenia is invariably present, and the blood platelet count may be reduced to 50,000/cu. mm. or lower. The tourniquet test produces multiple petechiae (Rumpel-Leede phenomenon). Bleeding time is prolonged, clotting time is normal, and clot retraction is delayed or absent. Prothrombin consumption is defective. Hyperplasia of the megakaryocytes of the bone marrow is always found, and erythroid hyperplasia is usually observed to a degree consistent with the anemia. The finding of marrow hyperplasia is an essential point in the differentiation of idiopathic from secondary purpura.

Spontaneous recovery is the rule in children; in this age group splenectomy should thus be reserved for uncontrollable bleeding and chronic, recurrent purpura which fails to respond to corticotropin or cortisone. In older patients the danger of intracranial hemorrhage is greater, and splenectomy is indicated if corticosteroids fail to induce a remission. Fresh blood, drawn within 6 hours in a plastic or siliconized container so as to preserve platelets, is administered preoperatively to correct hypovolemia, anemia, and thrombocytopenia.

Following splenectomy the response to corticotropin or cortisone may be improved even though thrombocytopenia may be incompletely corrected. Permanent remission follows removal of the spleen in 80-90% of cases.   •

SECONDARY HYPERSPLENISM

The same cytopenias caused by primary hypersplenism may also be produced by secondary hypersplenism. The splenic dysfunction and enlargement in secondary hypersplenism, however, are caused by chronic diseases such as leukemia and other lymphomas, tuberculosis and other chronic infections, sarcoidosis, Gaucher's disease, and congestive splenomegaly.

Hemolytic anemia is the most common form of secondary hypersplenism. Secondary splenic neutropenia, secondary splenic

panhematopenia, and secondary thrombocytopenic purpura also occur.

Although splenectomy does not affect the outcome of the chronic disease which is responsible for the secondary hypersplenism, the hematologic improvement which follows splenectomy justifies surgery in selected cases when other forms of therapy fail.

ACQUIRED HEMOLYTIC ANEMIA

There are 2 types of acquired hemolytic anemia: (1) idiopathic, in which autoantibodies of the agglutinin or hemolysin type are consistently found and the Coombs (human antiglobulin) test is positive; and (2) symptomatic, which is secondary to poisoning by some toxic agent (e.g., lead, sulfonamides) or malignancy (especially lymphoma). In the idiopathic variety of acquired hemolytic anemia spontaneous remissions may occur, and the manifestations can sometimes be controlled with corticotropin or cortisone. If corticosteroids are not effective, splenectomy should be considered although it is successful in only about 40% of cases. In the symptomatic type of acquired hemolytic anemia, splenectomy may be helpful when there is no response to treatment of the underlying disease or to the administration of corticosteroids.

ABDOMINAL HERNIAS

The following types of hernia involve the abdominal organs: (1) External abdominal hernia is any abnormal protrusion through the walls of the peritoneal cavity. This type of hernia presents to the outside, where it is usually visible and palpable. (2) Internal hernia is a protrusion of abdominal viscera into 1 of the internal abdominal fossae. (3) Diaphragmatic hernia is a protrusion of abdominal contents through a normal or abnormal opening in the diaphragm into the chest.

Hernias may be congenital or acquired. Acquired hernias are the result of trauma, increased intra-abdominal pressure, or abnormal relaxation of normal structures. A hernia may be reducible or irreducible (incarcerated). A strangulated hernia is an irreducible hernia whose blood supply is obstructed, so that the contents will become gangrenous within a few hours unless reduced. The complications of hernia consist of strangulation and the functional disturbances secondary to protrusion or incarceration. Because of the high incidence and possible serious consequences of these complications, surgical repair of most hernias is advisable.

EXTERNAL ABDOMINAL HERNIA

The relative incidence of external hernias is indirect inguinal (60%), direct inguinal (15%), umbilical (9%), incisional (9%), femoral (3%), epigastric (2%), and all others (2%). Those illnesses which cause intermittent or chronic increase of intra-abdominal

pressure, such as persistent cough or bladder neck or intestinal obstruction, should be sought for in older patients who develop or suddenly incarcerate a hernia. The treatment of external hernia is surgical. Trusses, binders, and belts are palliative measures reserved for the occasional patient who refuses or is unable to withstand operation.

Indirect Inguinal Hernia.

An indirect hernia consists of the protrusion of abdominal contents through the internal inguinal ring into a congenital diverticulum of peritoneum. This peritoneal sac is the unobliterated processus vaginalis which accompanies the descent of the testis in the male and fixation of the ovary in the female. The sac may be short or may extend into the scrotum to communicate with the tunica vaginalis of the testis. Although indirect hernia may occur at any age, it is more prevalent in the younger age group. There are 2 peaks of incidence of indirect hernia: during the first year of life and during the ages between 10 and 30. It is 9 times more common in males than in females.

A soft, nontender mass in the groin is the usual presenting complaint. Characteristically, this disappears on lying down and reappears on standing, or may be forced out by coughing or straining. Local burning or aching is common. Indirect hernias may extend to the bottom of the scrotum, but direct hernias rarely do so. Occasionally, a hernia is noted for the first time after an episode of sudden strain or severe coughing.

The hernia is best demonstrated by examination of the patient in the standing position. The scrotal skin is invaginated with the index finger until the fingertip is within the external inguinal ring. With the hernia reduced, a sudden cough by the patient will then force the hernia against or past the examining finger and demonstrate its oblique course down the length of the inguinal canal. Digital pressure over the internal inguinal ring will prevent the descent of an indirect hernia, whereas the bulge will still be felt if the hernia is direct. It is often impossible on physical examination to distinguish between direct and indirect hernia, and for practical purposes the distinction is of little importance. Hernias are often bilateral, and (especially in the older age group) direct and indirect hernias ("pantaloon" hernias) may occur together. Indirect hernias are occasionally "sliding," i.e., a viscus (usually the colon) forms part of the wall of the hernial sac. This type of hernia is frequently large and difficult to reduce.

The disorders most likely to be confused with hernia are hydrocele (distinguished by transillumination), varicocele, and inguinal nodes.

Surgical repair is the only satisfactory treatment, and can be done with minimal risk at all ages. Hernias should be repaired unless the patient's general health or advanced age make operation inadvisable. Under these circumstances symptoms can usually be controlled by a properly fitting truss. When a hernia is strangulated, as indicated by incarceration, local pain, and signs of intestinal obstruction, emergency operation is necessary. Following hernia repair the patient is restricted to light activity for 6 weeks to 3 months, but in the average case no restrictions of any kind need be imposed thereafter. The recurrence rate after indirect

inguinal hernioplasty is less than 1% in the hands of experienced surgeons. Higher rates are reported in many series.

Direct Inguinal Hernia.

The symptoms, signs, and treatment of direct inguinal hernia are essentially the same as in indirect hernia. Direct hernia often appears as a globular bulge in the region of the external inguinal ring and reduces immediately on recumbency. Direct hernias are rare in females. The tissues involved in direct hernia are usually less suitable for repair than in indirect hernia, and recurrence rates are generally higher (8-50%). However, when careful anatomic repair is carried out, the recurrence rate can be reduced to 1-5%.

Femoral Hernia.

A femoral hernia passes through the femoral ring and down the femoral canal to become subcutaneous in the fossa ovalis. It is more common in persons of middle age and is 4 times as frequent in females. These hernias are frequently overlooked, especially in obese individuals, because of their small size. The neck of the femoral sac is invariably small, and incarceration or strangulation of its contents is therefore not unusual.

The diagnosis of femoral hernia should always be considered in unexplained intestinal obstruction. Urinary symptoms, such as dysuria, frequency, or hematuria, will occasionally precede by several months any palpable evidence of a femoral hernia.

Differentiation from inguinal hernia can usually be made with ease by noting the anatomic landmarks. Femoral lymphadenitis may be difficult to distinguish from a strangulated femoral hernia because a tender mass is typical of both. An inflammatory lesion in the lower extremity, genitalia, or perineal region should be sought as a possible cause of lymphadenitis.

Surgical repair is the treatment.

Umbilical Hernia

Adult umbilical hernia is not uncommon, but occurs less frequently than the infantile type (see p. 161). The diagnosis is made with ease by inspection and palpation. The fully developed adult umbilical hernia is prone to incarceration and strangulation and should be repaired surgically.

Epigastric Hernia.

These hernias occur almost entirely in the linea alba between the xiphoid and the umbilicus. The hernia consists of a small mass of fat from the falciform ligament of the liver. Local discomfort is usually present even with very small hernias, and pain secondary to the hernia may be mistaken for that of peptic ulcer or cholelithiasis. Most of these hernias are irreducible and tender. Treatment is by surgical repair.

Incisional Hernia.

Incisional hernias are as various as the abdominal wounds from which they arise, usually as a result of inadequate closure, dehiscence, or infection. Symptoms consist of protrusion and discomfort. Because these hernias tend to increase in size and become more

difficult to manage, they should be repaired when the diagnosis is made.

Pelvic Hernia.

Hernias of the pelvic wall and floor are very rare. Obturator hernia leaves the pelvis through the obturator canal; sciatic hernia through the greater sciatic notch; pudendal hernia through the inferior sciatic notch; and perineal hernias through the pelvic diaphragm. These hernias are rarely palpable externally, and are of importance only as unusual causes of intestinal obstruction.

Other External Abdominal Hernias.

Semilunar (Spiegel's) hernia is a protrusion of preperitoneal fat between the aponeurotic fibers of the transversus abdominis, usually at the level of the linea semicircularis. These are very rare and are easily repaired.

A lumbar hernia may occur (1) through the inferior lumbar triangle of Petit, which lies just above the superior margin of the posterior crest of the ilium; or (2) through the superior lumbar triangle of Grynfelt, which lies just below the distal end of the twelfth rib. These hernias probably begin with the protrusion of retroperitoneal fat into a small neurovascular foramen. They are rare, but must be kept in mind as a possible cause of a mass or pain in the lumbar region. Surgical repair is the treatment.

INTERNAL HERNIA

Internal hernias are very rare. Herniation may occur through the foramen of Winslow or into the paraduodenal or other deep peritoneal recess. A traumatic rent in the omentum or mesentery may provide the hernial ring. Internal hernia must occasionally be considered in the differential diagnosis of intestinal obstruction.

DIAPHRAGMATIC HERNIA

A diaphragmatic hernia consists of the protrusion of 1 or more abdominal viscera into the thoracic cavity through an aperture in the diaphragm. Large congenital defects of the diaphragm with massive herniation of abdominal viscera usually cause severe symptoms in newborns and require immediate operation (see p. 150). Smaller congenital defects of the parasternal, pleuroperitoneal, or esophageal hiatal region may be the cause of symptomatic hernias in adults or may be incidental findings on x-ray. Clinically, the most frequent and important diaphragmatic hernias are the hiatal and the traumatic.

Esophageal Hiatus Hernia.

The common diaphragmatic hernia seen in adults is associated with an enlarged esophageal hiatus. There are 2 common types: (1) the sliding hernia, in which the cardio-esophageal junction moves upward into the chest, pushing the lower esophagus before it; and (2) the para-esophageal hernia, in which the cardio-esophageal junction tends to remain at its normal level while a portion of

the fundus herniates into the thorax alongside the esophagus. A very rare type of hernia is associated with a congenitally short esophagus in which the stomach extends upward through the hiatal opening to join the shortened esophagus in the chest.

Many of these hernias are asymptomatic and are discovered incidentally. The commonest symptoms are probably related to esophagitis and consist of substernal or epigastric distress and heartburn. Discomfort is often made worse by lying down after eating. Nausea and vomiting and dysphagia occur occasionally. Significant bleeding, noted in about 30% of cases, may be either massive or continuous, and may result in hypochromic anemia.

Physical examination is negative.

Barium study of the upper gastrointestinal tract establishes the diagnosis. Esophagoscopy is often indicated to determine the presence and degree of esophagitis.

Peptic ulcer, chronic cholecystitis, intra-abdominal malignancy, and coronary artery disease must be considered in the differential diagnosis. One must beware of attributing symptoms of a more serious underlying disorder to a hiatus hernia which is found incidentally on upper gastrointestinal x-rays.

Asymptomatic hernias require no treatment. Small hernias with mild symptoms usually respond to an ulcer-type regimen consisting of bland diet (see p. 94), antacids (see p. 612), and anticholinergic drugs (see p. 612). The patient should avoid lying down after meals and should sleep with the head of his bed elevated to minimize the reflux of gastric secretions into the esophagus. When there is a history of significant bleeding or when symptoms persist in spite of conservative measures, repair of the hernia by either the abdominal or the thoracic approach should be recommended.

Traumatic Diaphragmatic Hernia.

Most traumatic hernias involve the tendinous dome of the left diaphragm because the right lobe of the liver tends to protect the right leaf and seal any defects which occur there. Hernias may result from either penetrating or crushing injuries. Traumatic hernias do not have a peritoneal sac. Symptoms usually develop immediately after trauma, but in some instances may not appear for years. Complaints are chiefly respiratory or gastrointestinal, resulting from pulmonary compression or alimentary obstruction.

Diagnosis is established by x-ray.

Treatment is by reduction of the hernia and repair of the defect.

13 . . .

Gynecology

EXAMINATION OF THE GYNECOLOGIC PATIENT

A complete history should be taken on all gynecologic patients. Particular emphasis should be placed on menstrual history, past pregnancies, past gynecologic disorders, and the presence or absence of vaginal discharge, or pelvic pain.

A complete physical examination should also be done, with special attention to the vaginal and combined rectovaginal examination.

Besides the routine blood and urine examination the following tests and examinations are of value:

Gynecologic Laboratory Tests.
A. Vaginal Discharge: Hanging drop or direct saline microscopic examination for Trichomonas vaginalis and (with a few drops of 10% KOH added to clear the debris) for yeast cells.
B. Exfoliative Cytology: (See also p. 351.) Exfoliated material in the posterior vaginal fornix is collected, smeared on a slide, and fixed immediately. The interpretation of the stained slide for the determination of malignant cells or endocrine status requires special training.
C. Tissue Biopsy: Varies from biopsy of a cervical lesion in the office to endometrial biopsy via dilatation and curettage in the operating room.
D. Tests of Endocrine Function: These are used to test hormonal function and include vaginal cell cornification, the cervical smear fern test, and biologic tests for pregnancy or hormonal titers.

LEUKORRHEA

Leukorrhea may be defined as a nonbloody discharge from the vagina, usually white but at times yellowish. Normal vaginal "discharge" is clear or white (due to the presence of desquamated epithelium) and is normally present in an amount sufficient to lubricate the vaginal walls; occasionally there may be a small amount of overflow, usually at the time of ovulation.

Trichomonas vaginalis Vaginitis.
Trichomonas vaginalis causes a vaginal discharge which is heavy, yellow, nonviscous, frothy, has an offensive smell, and is associated with vulval itching. The vaginal mucosa is erythematous and dotted with small areas averaging 5 mm. in diameter which are more red than the surrounding tissue ("strawberry mucosa"). The diagnosis is made by inspecting a small amount of the

discharge microscopically.

About 90% of cases will respond to 1 course of trichomonacide treatment. In stubborn cases the organism may be found in the bladder or in the deep cervical mucus glands; conization of the cervix may prove to be beneficial in the latter case.

Fungal Vaginitis.

Mycotic vaginitis is caused by yeast-like organisms which are often found normally in small numbers in the vagina but which tend to overgrow following antibiotic therapy, in diabetes mellitus, or spontaneously.

The mucosa is intensely inflamed, and the discharge is "cheese-like" in masses of white material. Microscopic examination of a sample of this vaginal discharge shows mycelia and conidia.

The treatment of mycotic vaginitis is usually simpler than that of trichomonal infection. Two to 4 thorough paintings of the vagina and vulva with a 2% aqueous solution of gentian violet or nightly insertion of 1 nystatin (Mycostatin®) vaginal tablet following a vinegar douche for 15 nights will usually effect a cure. Propion-Gel® inserted vaginally, following a douche, each night for approximately 3 weeks, will cure most cases also.

Noninfectious Leukorrhea.

Leukorrhea at times takes the form of profuse clear mucus which is obviously not infected. This is usually due to a chronically irritated cervix, especially in the endocervical canal.

Treatment is by cauterization of the cervix with silver nitrate or with the electrocautery unit. Conization of the cervix may be necessary to decrease the number of mucus-producing glands.

Bacterial Leukorrhea.

Simple bacterial invasion of the intact vagina does not itself cause leukorrhea since the normal nonpathogenic flora will usually overwhelm any pathogens which gain entrance. However, an infected lesion of the vagina or cervix may cause leukorrhea. Specific antibiotics are indicated.

Miscellaneous Rare Causes of Leukorrhea.

Endometritis and fallopian tube inflammation or malignancy may cause leukorrhea. An excess of normal mucus may occur with chronic pelvic congestion or in association with emotional disorders.

Gonorrheal Leukorrhea in Children.

Gonorrheal vulvovaginitis occurs only in children, since the mature vaginal epithelium seems to resist infection by this organism. Treatment is by specific antibiotics.

DISORDERS OF THE EXTERNAL
FEMALE GENITALIA

IMPERFORATE HYMEN

Imperforate hymen is a rare disorder which may escape detection until the menarche. The onset of menstruation without an avenue of egress for the blood causes distention of the vagina (hematocolpos) and, if not relieved, distention also of the cervix (hematotrachelos), the corpus uteri (hematometra), and the fallopian tubes (hematosalpinx). The symptoms are pain, increasing with each episode of endometrial desquamation; the absence of external bleeding; and, in extreme cases, lower abdominal swelling. Diagnosis is made by finding the bulging, intact hymen. Treatment consists of simple incision of the hymen, with revision to prevent recurrence.

KRAUROSIS VULVAE

Kraurosis vulvae is a degeneration of the subcutaneous fat of the vulvar area, resulting in stretching and thinning of the overlying skin. Medical treatment consists of (1) local estrogen therapy in the form of cream or as a vaginal suppository inserted nightly; and (2) vitamin A, 50,000 units t.i.d. orally for several months. Surgical treatment consists usually of plastic alteration of the introitus to overcome dyspareunia. Vulvectomy may be necessary in extreme cases (see Leukoplakic Vulvitis, below).

LEUKOPLAKIC VULVITIS

Leukoplakic vulvitis is a chronic disorder of unknown cause affecting part or all of the vulva and characterized by white plaques of parchment-like skin. It usually seems (like kraurosis) to be associated with a lack of estrogen and so is most commonly seen in postmenopausal women. The symptoms are pruritus, burning, and dyspareunia.

Leukoplakic vulvitis should be considered a precancerous lesion, since about half of all cases of carcinoma of the vulva occur in an area of leukoplakia. For this reason no treatment other than surgical excision can be advocated. A biopsy should be performed prior to surgery; if malignancy is present, radical vulvectomy and lymph node excision should be performed. Leukoplakia may recur.

BENIGN NEOPLASMS
OF THE EXTERNAL GENITALIA AND VAGINA

Cysts.
Sebaceous cysts of the vulvar area are quite common. They may be as large as 4-5 cm. in diameter (usually smaller), are firmly cystic, and are fixed to surrounding tissue. They are not tender unless bruised or secondarily infected. Treatment is by

extirpation or by marsupialization and silver nitrate cauterization of the cyst wall.

Wolffian duct cysts are congenital remnants which are usually found in the anterior lateral portion of the vulva (especially near the clitoris). They are typically small, thin-walled, and pedunculated. Treatment is by surgical removal.

Solid Tumors.

Solid benign tumors of the vulva may be composed of any tissue found in the area (leiomyoma, fibroma, lipoma, angioma, hidradenoma, or papilloma). A presumptive diagnosis can be made by inspection of the tumor. Histologic examination of the excised tumor (or biopsy, if malignancy is suspected) will lead to definitive diagnosis.

Condyloma acuminata is a rather common papillomatous growth of epithelium which varies in size from a small "wart" to an extensive distribution of isolated growths or a confluent mass covering the entire vulvar, perineal, anal, and upper thigh areas. It is sometimes seen also in the vagina and on the epithelium of the cervix. Originally termed "venereal wart" because it commonly coexists with gonorrhea, it may occur as a result of chronic local irritation due to persistent vaginal discharge from any cause.

Microscopically, the tumor is a papilloma with great masses of stratified squamous epithelium (often in whorls, with central degeneration), giving an epithelial pearl-like appearance. The firm basement membrane which is always present differentiates this lesion from malignant papilloma.

The lesions may be extirpated surgically or destroyed by electrocoagulation. Some success is reported with local application of podophyllin, 20-25% in an oil or ointment base, for 8 hours. If the original source of vulvar irritation is not removed, the lesions are apt to recur.

MALIGNANT NEOPLASMS
OF THE EXTERNAL GENITALIA AND VAGINA

Carcinoma of the Vulva.

Carcinoma of the vulva may be secondary to a primary lesion of 1 of the other genital structures, but this is rare. Primary carcinoma of the vulva, comprising 4-5% of all gynecologic cancers, is usually found in women 60-70 years of age. Diagnosis is not difficult since inspection of the vulva reveals the lesion and a biopsy specimen for histologic examination can be quickly obtained. The neoplasm is nearly always epidermoid, and may display any degree of anaplastic activity.

Since half of all cases of vulvar carcinoma are preceded by leukoplakia, the most important aspect of treatment is prophylactic removal of leukoplakia. The treatment of vulvar carcinoma is radical surgical removal (including all regional lymph nodes), since the lesion has usually proved to be resistant to irradiation. Operative treatment is often remarkably successful; five-year survival rates as high as 90% have been reported.

DISORDERS OF THE FEMALE URETHRA

INFLAMMATORY URETHRAL DISORDERS

The para-urethral glands (Skene's) may become abscessed (often as a result of gonorrheal infection), and should be drained, if possible, by locating and opening the ducts through the urethral mucosa. This disorder may be simulated by a urethral diverticulum (which may have been formed by a previous abscess). The diverticulum may be diagnosed by endoscopic examination or x-ray films using contrast media. Treatment of diverticulum is by surgical excision and reconstruction.

PROLAPSE OF THE URETHRA

Prolapse of the urethra resembles and may be difficult to differentiate from neoplastic tissue. Reduction may be attempted, but resection must be resorted to if this is not successful. Since the prolapsed mucosa may bring the ureteral orifices down with it, urography should be performed before resection.

NEOPLASMS OF THE URETHRA

Neoplasms are occasionally seen in the urethra. Caruncle, usually seen in women past the menopause, is a benign tumor of the urethral meatus which presents as a red, raspberry-like tumor. It is quite painful and tender. Diagnosis depends upon microscopic examinations of an excised specimen. There are 3 histologic types: papillomatous, angiomatous, and granulomatous. Treatment is by resection, which may be technically difficult because of the great vascularity of the region.

Carcinoma of the urethra is uncommon and sarcoma is rare. They are differentiated from caruncle by biopsy. Treatment is by radical surgical extirpation unless the lesion has progressed to the incurable stage, in which case palliative irradiation may be useful.

BARTHOLIN'S CYST AND ABSCESS*

A cyst of a Bartholin's gland may develop following infection as a result of obstruction of the draining duct by scarring. An uninfected cyst is not harmful, but it may be annoying to the patient. Aspiration is rarely successful; surgical excision or marsupialization is usually necessary.

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*Carcinoma (adenocarcinoma) of Bartholin's glands is occasionally seen. An epidermoid form may occur which most likely originates in the transitional epithelium of the duct. The carcinoma presents as a localized mass which is painless unless secondary infection occurs. Treatment consists of radical bilateral vulvectomy and inguinal node resection.

DISORDERS OF THE VAGINA

CONGENITAL DISORDERS OF THE VAGINA

If congenital vaginal septa or constricting bands interfere with coitus or childbirth, plastic repair is indicated. More serious congenital disorders such as total absence of the vagina are rarely encountered. Proper management includes accurate diagnostic measures to ascertain the patient's genetic sex; psychiatric evaluation of the patient's libido (psychic sex); and often extensive and expert plastic surgical procedures.

INJURIES TO THE VAGINA

Traumatic lesions of the vagina are most commonly caused by the instrumentation involved in attempted criminal abortion. The inept abortionist (often the patient herself) may force an instrument into the vagina as far as possible, thus injuring or perforating the posterior fornix. If perforation has occurred, exploratory laparotomy may be indicated to rule out injury, infection, or chemical irritation. Suturing usually suffices to repair the defect.

In some cases of attempted abortion the vaginal lesion may be a caustic burn caused by the insertion of potassium permanganate tablets or other chemicals. These usually burn a hole in the mucosa which may penetrate up to 2 cm. in depth. Bleeding may be brisk, but can be controlled by suturing.

NEOPLASMS OF THE VAGINA

Gartner's duct cysts arise in unobliterated remnants of the lower portion of the wolffian duct, and thus are seen only in the anterolateral portion of the vagina. These cysts are usually small and asymptomatic, but may become large enough to cause dyspareunia. Treatment is by surgical removal. Microscopic examination of the cyst wall shows the cuboidal ciliated epithelium of the wolffian remnant, which may be flattened by the internal pressure of the cyst.

Inclusion cysts may be formed by infolding of the epithelial surface following obstetric or surgical tissue disruption. The infolded mucosa desquamates, producing a cyst of thick white material. These cysts are rarely large or symptomatic, but they may be excised easily if annoying.

The relatively rare solid benign tumors of the vagina are composed of any of the tissues usually found in the area: myoma, fibroma, papilloma, and myxoma. If there are no symptoms, surgical removal is indicated only to rule out malignancy by histologic examination of the specimen.

Primary carcinoma or sarcoma is rarely seen. Bleeding, especially postcoital, is the presenting sign. Biopsy is required. Lymphatic spread is early and rapid, and local extensions often cause fistulas. The survival rates following radical surgical pro-

cedures have not been encouraging; the usual method of treatment is irradiation, but the five-year survival rate is only about 20%.

DISORDERS OF THE CERVIX

CONGENITAL LESIONS OF THE CERVIX

Congenital atresia or even absence of the cervix may occur, but congenital cervical lesions are usually associated with abnormalities of the corpus of the uterus and will be considered in that section.

TRAUMA TO THE CERVIX

Traumatic cervical lesions are nearly always the result of instrumentation or childbirth. In the former case the damage can be seen by inspection and appropriate surgical repair instituted. The bilateral cervical tearing associated with 1 or more deliveries tends to change the cervical os from a small round opening 5 mm. in diameter to a transverse opening. These tears may be so deep that anterior and posterior "lips" gape open and expose the mucosa of the cervical canal. This tends to cause an excess of mucus production, usually the only symptom. Moderate lesions can be treated in the office by cauterization of the angles of the cervix. More extensive disruptions will require plastic surgical repair.

Incompetent internal cervical os is a disorder in which the internal os fails to hold a pregnancy, giving way in the second trimester or early in the third trimester. It may be congenital, but is usually due to the trauma of a previous delivery or a too forceful instrumental cervical dilatation. The incompetence of the os can be demonstrated, after complete postpartum uterine involution, if a sound or dilator more than 7 mm. in diameter can be passed into the canal. The defect may also be demonstrated by radiographic means, but this is rarely necessary.

Treatment consists of either (1) resecting a portion of the cervix at the level of the internal os and closing with sutures, or (2) strengthening the tissue in the area by the deposition of irritants for the purpose of producing fibrosis. The fetal survival rate in future pregnancies (following treatment by either method) is about 50%; as experience with the irritant method accumulates, the prognosis should improve.

INFLAMMATORY DISORDERS OF THE CERVIX

Inflammation of the cervix may be acute, as in acute gonorrhea, but this is quite rare. Chronic nonspecific cervicitis, however, is one of the most common lesions seen in the gynecologist's office. The chronically irritated tissue of the portio vaginalis area surrounding the external cervical os, normally covered by stratified squamous epithelium, becomes overgrown by the cylindric mucus-

producing cells of the cervical canal, producing the 2 character-
istics of the lesion: leukorrhea and a reddened surface which looks
like an erosion. If the lumens of the mucus-producing glands of
the cervix become occluded, retention cysts (nabothian cysts) are
found. Microscopically, the submucosal connective tissue is infil-
trated by round cells, plasma cells, and sometimes by polymorpho-
nuclear cells. The gland epithelium is often replaced in some areas
by a stratified squamous epithelium (epidermidalization) which may
be difficult to distinguish from carcinoma.

Mild cervicitis may be treated by local caustic (silver nitrate)
applications, carefully applied. The usual method of treatment,
however, is destruction of the overgrown cylindric epithelium by
electrocautery so that a normal squamous epithelium can develop.
Cauterization can be performed in the office without anesthesia
since the cervix has few nerve-ends sensitive to heat. The cautery
tip is stroked over the surface of the cervix in a radial pattern and
may be inserted 1-2 cm. into the cervical canal if necessary. Light
pressure on the cautery tip is usually advisable since the lesion is
rarely deep in the tissues. Cauterization of the endocervix should
be judiciously performed because excessive scar tissue might cause
stenosis of the cervix. Stenosis can be prevented by monthly pas-
sage of a uterine sound until patency of the cervical canal is assured.

Other inflammatory lesions of the cervix such as tuberculosis
or the granulomatous venereal diseases occur rarely. Diagnosis
depends upon biopsy and specific tests.

Stricture of the cervix may follow inflammation or the treat-
ment of inflammation, but since stricture may also be caused by
tissue proliferation malignancy must be ruled out. During the
menacme the symptoms are lower abdominal pain and cramping
due to hematometra; the postmenopausal patient may have the same
symptoms plus fever (due to pyometra) or may have no symptoms.
Treatment is by dilatation, which may be sufficiently difficult to
warrant hospitalization and anesthesia.

Leukoplakia of the cervix, not often seen, is considered by
some to be the same precancerous lesion as leukoplakia of the vul-
va (see p. 345), but this view is not supported by statistics. Even
so, the lesion should be biopsied and, if not malignant, observed
repeatedly for evidence of malignant change.

CERVICAL POLYP

The most common benign neoplasm of the cervix is the cervical
polyp, a localized area of endocervical epithelium which enlarges
until it becomes pedunculated. Polyps may be single or multiple,
and vary from 1-2 mm. to 7-8 cm. in diameter. Contact bleeding
(coitus, douching) commonly occurs, and there may be an excessive
nonbloody discharge.

The polyp should be removed, usually by simply twisting it off
and lightly cauterizing its base. If numerous polyps are seen high
in the cervical canal, it may be necessary to dilate and curet the
canal under anesthesia. The polyps should be examined micro-
scopically for malignancy.

CERVICAL CARCINOMA

Carcinoma of the cervix is the commonest (55%) of all female pelvic malignancies. Two to 3% of all cervices become carcinomatous, usually in patients between the ages of 45 and 55. Recent evidence suggests that cervical carcinoma is more common in women who have had prolonged exposure to the smegma bacillus (those who marry while quite young) and who have had several children than in women who have had no contact with smegma (virgins, Jewish women) or have had no children.

The carcinoma may be adenomatous (5%) or epidermoid (95%); the gross appearance, symptoms, and management do not differ in the 2 types.

Clinical Findings.
  A. Symptoms: The symptoms, in the usual order of appearance, are abnormal discharge, bleeding (especially contact), and, in the later stages, pain. Most other symptoms are the result of extension of the disease.
  B. Signs: Inspection usually discloses an eroding (or, at times, benign-appearing) lesion which bleeds easily, but there may be no visible lesion. On palpation the cervix usually has a hard consistency; it may or may not be nodular. In the later stages of the disease, extension of the mass may be felt as it grows into the parametrial area or the vagina, bladder, or rectum.
  C. Laboratory Findings: The diagnosis may be suggested by the vaginal "smear test," but must be proved by biopsy.
    1. The Papanicolaou smear test consists of smearing a slide with desquamated cervical epithelial cells found in the vaginal fornix and staining and examining the cells for malignancy. Several methods of preparing the slides have been developed, and various systems of reporting the findings are in use; it is important for each physician to prepare his slides in the manner advised by his cytologist and to have a complete understanding of the significance of the cytologist's report.

       The Papanicolaou smear should be part of the routine examination of women in the cancer age group. It is considered accurate in 95-98% of cases of carcinoma of the cervix and in 60-90% of cases of carcinoma of the corpus, but it is unreliable in carcinoma of the tube and of no value in the diagnosis of ovarian malignancy. In any case it is a screening test only, to be verified by biopsy if positive.

       The technic of taking and mailing Papanicolaou smears is described in "Cytology and Cancer of the Cervix," American Cancer Society, 1957.
    2. Biopsy of the cervix may be done by the use of a punch biopsy forceps specially designed for the purpose or by knife or scissors excision of the tissue. A specimen should be obtained from each of the 4 quadrants of the cervix. It is often preferable to do a conization of the cervix, particularly if no definite lesion is visible. By whatever method the biopsy is taken, adequate tissue is essential and the area of the squamo-columnar junction must be represented.

Clinical Classification.

Once the diagnosis is made, the carcinoma must then be "staged." Since the method of treatment employed varies with the degree of extension of the malignancy and since the comparison of results obtained by various methods depends upon an agreed terminology, a classification into clinical "stages" has been developed The International Classification, an outgrowth of the League of Nations Classification, is now used almost exclusively. It is as follows:

Stage 0: Carcinoma in situ (also termed intraepithelial carcinoma or preinvasive carcinoma). In Stage 0 the carcinoma has not extended past the basement membrane of the epithelium.

Stage I: The carcinoma has extended past the basement membrane but is confined to the cervix.

Stage II: The carcinoma extends past the cervix into the parametrial tissues or the vagina, but has not reached the pelvic wall or the lower third of the vagina.

Stage III: The carcinoma has reached the pelvic wall or the lower third of the vagina.

Stage IV: The carcinoma has extended into the rectum, the bladder, or any other area beyond those previously defined.

The "stage" is properly included in the disease nomenclature, e.g., "carcinoma, epidermoid, of cervix, Stage II."

Repeated attempts have been made to "grade" the tumor according to its microscopic appearance, with the object of basing the selection of methods of treatment on the degree of anaplasticity of the cells. No practical application of this categorization has as yet proved of value.

Treatment.

A. Stage 0: Stage 0 is usually treated by total hysterectomy, but this degree of malignancy does not necessitate removal of the adnexa. Cervical amputation or conization may be adequate to remove an intraepithelial lesion, and this might be considered should the patient desire future pregnancy. However, she should be made aware of the possible inadequacy of this treatment and should be advised to return at frequent intervals for visual and cytohistologic examination.

B. Stages I-IV: When adequate facilities and personnel are available, radium and/or external irradiation therapy is usually preferred. The best method of radiation therapy is to deliver a minimum of 6000 r to the cervix and the areas of lymphatic spread at the sides of the cervix. However, some gynecologists prefer radical pelvic surgery: extirpation of the uterus with a cuff of vagina, tubes, ovaries, the regional lymph nodes, and all intervening tissue which may contain channels of lymphatic spread or points of direct extension. The surgical approach is generally used in situations where radiotherapy has been tried without complete success or where surgical personnel have demonstrated their ability to carry out this radical procedure without a prohibitive operative mortality rate.

Prognosis.

Using the best methods of treatment available at this time, the following five-year survival rates are to be expected:

Stage 0: 100%
Stage I: 70-90%
Stage II: 35-60%
Stage III: 5-15%
Stage IV: 0%

CERVICAL SARCOMA

Botryoid ("grape-like") sarcoma is a rare dysontogenetic cervical lesion which is usually found in early life (at times even at birth) or after the menopause. It presents as a mass of red or purplish polypoid structures, which may fill the vagina. Histologically the tumor is composed of various embryonic tissues (e.g., striped muscle, cartilage, nerve tissue) in the mass of malignant connective tissue cells. Surgical removal and/or irradiation may be employed in treatment, but cures are rarely reported.

Sarcoma of the cervix composed of round or spindle cells has been reported, but these are so extremely rare that they will not be discussed here.

DISORDERS OF THE CORPUS UTERI

CONGENITAL LESIONS OF THE UTERUS

Congenital lesions of the uterus are not uncommon; some type occurs in 1-2% of all women. The anomaly is due to failure of complete fusion of the two müllerian ducts, which produces various degrees of reduplication of the genital structures. Failure of development of one müllerian duct results in uterus unicornis or uterus bicornis unicollis with one horn rudimentary. Failure of development of both müllerian ducts results in hypoplasia of the uterus, varying in degree from minor (infantile uterus) to marked (absence of the uterus).

The clinical significance of these disorders varies with the type of anomaly: amenorrhea with absence or marked hypoplasia of the uterus, relative or absolute infertility, and interference with the development of a pregnancy or dystocia at the time of delivery. Many patients, however, achieve conception and uncomplicated parturition despite anomalous genitalia.

Plastic surgical procedures on uteri of adequate size are often successful in correcting infertility or dystocia. Hormonal treatment of uterine hypoplasia has been disappointing.

TRAUMATIC DISORDERS OF THE UTERUS

Perforation of the wall of the uterus during dilatation and curettage is the only significant uterine trauma of appreciable incidence. If, during this procedure, the instrument should penetrate the wall and the patient goes into shock, immediate laparotomy for the purpose of terminating hemorrhage and repairing or removing the damaged organ is indicated. If, as is more often the case, the perforation does not produce untoward symptoms, observation is all that may be required.

MYOMAS OF THE UTERUS

Myomas of the uterus ("fibroids"), composed of smooth muscle cells of the uterine corpus, are found in about 20% of all white women over 30 years of age; colored races are more commonly afflicted. The etiology is not known, but since these tumors appear during the menacme and stop growing with the menopause, the ovarian hormones have been incriminated. Myomas are associated with infertility, but no pathogenetic relationship has been established.

Myomas can occur singly but are usually multiple. They vary in size from "seedlings" to massive growths filling the abdominal cavity and weighing up to 100 lb. According to their position in the uterine wall, these tumors may be characterized as submucous, intramural, or subserous (submucous and subserous myomas may be pedunculated). A myoma may also grow to the side of the uterus between the leaves of the broad ligament (intraligamentary myoma).

Clinical Findings.

   A. Symptoms: Many patients with uterine myomas have no symptoms even when the tumor is of considerable size; other women present only as infertility problems. Symptoms, when present, depend upon the size and position of the growths. Submucous myomas tend to cause abnormal bleeding by eroding the mucosal surface on the same or opposite side or by interfering with the normal vascular pattern of the overlying endometrium. If submucous myomas are large or pedunculated they may cause pain, especially of the dysmenorrhea type. Subserous myoma in itself causes no bleeding and no pain, but it may disrupt the menstrual pattern by interfering with normal ovarian function and may cause pain if it presses upon an adjacent structure. A pedunculated tumor may become twisted, precipitating an acute abdominal crisis.

   B. Signs: The physical examination will disclose a tumor of palpable size, but other firm pelvic tumors may also present similar pelvic masses. A submucous myoma would not be apparent to the examiner's hands; it may be suspected if the uterus is enlarged, if other types of myoma are present, or if an abnormal mass is felt in the uterine cavity with the probing tip of a uterine sound or curet.

Differential Diagnosis.

In the management of myomas of the uterus, it must be borne

in mind that bleeding from the uterus, even in the presence of my-
omas, may be due to other causes (specifically, carcinoma).

Treatment.

In most cases the best treatment is total hysterectomy. If the
patient desires future pregnancy and if the uterus is sufficiently
intact so that pregnancy may be anticipated, myomectomy may be
preferred. However, the pregnancy rate following myomectomy
is disappointingly low, and the recurrence rate due to growth of
unexcised "seedlings" is high. The growth of the tumor may be
halted by irradiation castration, but this method of treatment is not
commonly used today. Myomas which are not too large, are not
growing rapidly, and are asymptomatic may be kept under frequent
observation in the expectation that no complication will arise after
the menopause.

Course and Prognosis.

Degenerative changes may occur, usually as a result of alter-
ations in vascularity. The types of degeneration are (1) hyaline,
(2) cystic, (3) calcific, (4) suppurative, (5) necrotic ("red"), (6)
fatty, and (7) sarcomatous. The degeneration may cause pain due
to necrosis, but is otherwise of no clinical significance except for
the 0.5-1% which become sarcomatous (see Sarcoma, below).

ADENOMYOSIS OF THE UTERUS

Adenomyosis of the uterus is the extension into the myometrium
of benign endometrium. When extensive, the growths cause a dif-
fuse uterine enlargement which is rarely sufficient to be noticeable
upon bimanual pelvic examination. Microscopically, endometrial
tissue is scattered throughout the myometrium, and the extension of
this tissue from the surface of the uterine wall can sometimes be
traced. The symptoms are pain with menstruation and, in some
cases, increased menstrual flow. Bimanual examination, espe-
cially at the time of menses, discloses a tender, boggy uterus.

The only treatment is hysterectomy, if required.

CARCINOMA OF THE UTERUS

Carcinoma of the corpus uteri involves the endometrium. It
occurs almost as frequently as carcinoma of the cervix, usually in
the age group from 50 to 60. Histologically the tumor is usually an
adenocarcinoma, with marked overgrowth of endometrial tissue and
typical malignant cell changes. Occasional variations in appearance
(e.g., "squamous cell carcinoma") are seen. Growth of the tissue
into the uterine cavity eventually produces bleeding. Extension into
the wall of the uterus may be minimal or marked. Lymphatic or
hematogenous spread to other pelvic organs or distant sites is typi-
cally late.

The presenting symptom is nearly always abnormal bleeding;
the postmenopausal bleeder is particularly apt to have carcinoma.
There may be abnormal discharge, and pain is a late symptom.

Since excessive or prolonged exposure to estrogens is associated

with greater than normal incidence of uterine carcinoma, this tumor should be watched for especially in the patient with a history of late menopause, endometrial hyperplasia, or the diabetes-obesity-hypertension triad.

The definitive diagnosis can be made only by the microscopic examination of endometrium obtained by curettage or biopsy. At present the Papanicolaou smear test is not as reliable for corpus carcinoma as it is for cervical carcinoma.

Treatment and Prognosis.

Treatment consists of total hysterectomy and bilateral salpingo-oophorectomy; lymphadenectomy also may occasionally be indicated.

Some gynecologists prefer to irradiate the uterus prior to surgery in the belief that irradiation prevents spread of the tumor, but recent statistics indicate that this is not necessary since surgery alone has a five-year survival rate of up to 90%. If intercurrent disease or poor physical condition contraindicates surgery, intracavitary radium and external irradiation may be used; fairly good results (five-year survival rates up to 50%) have been reported.

SARCOMA OF THE UTERUS

Sarcoma may arise from any of the connective tissue elements of the uterus, but it most commonly originates in a myoma (0.5-1% of myomas). Abnormal vaginal discharge and bleeding are the only symptoms until the growth reaches a noticeable size or causes pressure pain in an adjacent structure; unusually rapid growth of a myoma should alert the physician to its presence. A negative Papanicolaou smear is unreliable as a diagnostic aid.

Treatment.

Treatment is by surgical removal of the entire uterus and both tubes and ovaries. Since sarcoma is usually not discovered until operation, it is essential that all myomas be bisected for inspection at this time so that the surgeon can remove all of the internal genital structures if the malignancy is discovered. If a report of sarcoma is not received from the pathologist until several days after supravaginal hysterectomy, the cervical stump should receive a course of irradiation, usually (and preferably) with radium. Supravaginal hysterectomy, however, is rarely indicated today. The total uterus should almost always be removed, and there can certainly be no justification for failing to remove the cervix in a postmenopausal patient with a growing tumor of the uterine corpus.

DISORDERS OF THE FALLOPIAN TUBES

ACUTE PELVIC INFLAMMATORY DISEASE

Infection of the fallopian tubes is extremely common during the menacme but rare in women who have ceased to menstruate. The most common infecting organisms are the gonococcus and strepto-

coccus, but staphylococci, Escherichia coli, and occasionally tubercle bacilli may also be found. Salpingitis may occur (independently of venereal infection) in the immediate postpartum period as puerperal sepsis and accompanying abortion as septic abortion.

In most instances of salpingitis the infection spreads rapidly throughout the pelvis ("pelvic inflammatory disease"). The contralateral tube is involved within 24 hours, and within 72 hours the infection will spread to the entire uterus, all adnexal structures, and the parametrial lymphatic, vascular, and areolar tissues. As the process continues there is infection of the adjacent visceral and parietal peritoneum. Pelvic thrombophlebitis is common, and embolism may occur. As the body defenses come into play, the infection will become walled off by the formation of abscesses. These may involve a tube (pyosalpinx) or a tube and ovary (tubo-ovarian abscess), or may lodge in the cul-de-sac as a "pelvic abscess." Eventually the abscesses become sterile, the purulent elements are absorbed, and the dilated tube - now a thin-walled sac - becomes filled with a clear, sterile fluid (hydrosalpinx). The pelvic abscess, if untreated, usually ruptures spontaneously into the posterior fornix of the vagina, occasionally into the abdominal cavity, or, more commonly, into the rectum or bladder.

Clinical Findings.

Inflammation causes pelvic pain, fever, chills and sweats. Vomiting, bloating, and constipation result as pelvic peritonitis develops. Pelvic examination reveals an excruciatingly tender pelvis, with a mass or masses if the walling off process has begun. If peritonitis has developed there will be a rigid abdomen, rebound tenderness, and decreased peristalsis. The WBC will be high with a marked shift to the left, and the sedimentation rate will be accelerated.

Treatment.

Medical treatment consists of appropriate antibiotics in adequate amounts, usually a broad-spectrum antibiotic or a combination of antibiotic drugs. Other measures include absolute bed rest and fluid replacement. Should a pelvic abscess "point" in the posterior vaginal fornix, the fluctuant, bulging area should be incised (colpotomy) and rubber drains placed in the cul-de-sac for 4-7 days.

If the infection can be halted by antibiotics before an abscess has formed, a definitive cure may be obtained.

For marked destruction of the reproductive organs ("pelvic cripple"), the usual surgical procedure is removal of both tubes and the uterus. An involved ovary should also be removed, but a normal ovary should be left in place unless there is some other reason for its removal.

Prognosis.

Before the era of antibiotic therapy the mortality rate of pelvic inflammatory disease was as high as 25% in some series. Although the mortality rate is now almost negligible, this is still a serious disease since it causes infertility and may become chronic with recurrent acute exacerbations (see below).

CHRONIC PELVIC INFLAMMATORY DISEASE

Chronic pelvic inflammatory disease is nearly always preceded by the acute disease. In many instances an acute attack which has apparently been cured by antibiotics will recur. The patient may be completely free of symptoms and abnormal physical findings except during repeated acute attacks, or there may be residual pelvic "soreness" between acute attacks which causes dysmenorrhea and dyspareunia and forces the patient to limit her activities ("pelvic cripple"). Although antibiotic therapy may be effective during an acute exacerbation, surgery is usually required (see above).

TUBERCULOUS SALPINGITIS

Tuberculosis of the pelvis (usually secondary to tuberculosis elsewhere) most commonly involves the fallopian tubes first and most extensively. Although tuberculous infection of the tubes has been reported in 5% of all cases of pelvic inflammatory disease, its incidence is decreasing. It rarely becomes acute, more often presenting the symptoms and signs of chronic pelvic inflammatory disease. Since it involves the tubes primarily, it is sometimes found in the patient whose only complaint is infertility.

The disease is diagnosed at surgery or in the pathology laboratory. The typical tubercles on the peritoneal surface of the tubes will, of course, indicate the true nature of the inflammatory process, but these are not commonly present.

In the past treatment has been surgical removal of all pelvic organs. However, antituberculosis drugs should be tried first in patients who desire future pregnancies.

NEOPLASTIC DISEASE OF THE FALLOPIAN TUBES

Carcinoma (adenocarcinoma) of the fallopian tubes is not common. Because it may not cause bleeding or desquamation of tumor cells to and through the uterus and cervix, the chances of making an early diagnosis on the basis of abnormal bleeding or a positive Papanicolaou smear are remote. The patient may notice gradually intensifying one-sided pelvic pain. A growing adnexal mass, especially in a postmenopausal patient, should be investigated surgically.

The only treatment is surgical removal, but the cure rate is only about 5-10% and most of these few successes are with early tumors discovered during surgery for some other condition.

Parovarian cysts (wolffian remnants) may develop in the mesosalpinx and may reach such a size that removal is indicated. It is not possible by pelvic examination to differentiate between an ovarian and a parovarian cyst. This is immaterial, however, since cysts of a certain size (see p. 359) must be removed.

Parovarian cysts are rarely malignant.

DISORDERS OF THE OVARIES

The ovaries may be small or absent as a result of congenital maldevelopment; and they may become involved in pelvic inflammatory disease (see p. 356). Aside from these abnormalities, ovarian disorders are almost exclusively neoplastic in origin.

OVARIAN TUMORS

Because ovarian tumors are so varied in type and because our knowledge of the origins of some of them is incomplete, it is not possible to develop a completely satisfactory system of classification. The classification used here is chosen primarily for its clinical usefulness.

Cystic Tumors.
  A. Nonneoplastic:
      1. Follicle cysts - These are caused by failure of the normal follicle to rupture or failure of the incompletely developed follicle to be reabsorbed. They rarely grow to more than 5 cm. in diameter or persist longer than 60 days; a larger or more persistent ovarian cyst should be considered pathologic. Symptoms are produced by follicle cysts only in the uncommon event of torsion or rupture with hemorrhage. The cysts are usually reabsorbed spontaneously.
      2. Corpus luteum cysts - Usually caused by an excess amount of bleeding into the corpus luteum cavity. This may be associated with pregnancy, and very large lutein cysts occasionally accompany hydatidiform mole. Corpus luteum cysts may cause a delay in menstruation; otherwise symptomatology and course are as described for follicle cysts.
      3. Germinal inclusion cysts - Incidental findings at microscopic examination of ovaries; of no clinical importance.
      4. Endometrial cysts - See Endometriosis, p. 365.
  B. Neoplastic:
      1. Benign -
         a. Pseudomucinous cystadenomas - These may become huge, are usually multilocular, and contain a clear, more or less viscid fluid. They are lined by tall columnar cells, and goblet cells are usually present also. This suggests the most commonly accepted theory of their histogenesis: that these cysts are teratomas composed entirely of entoderm. Some may originate in Brenner tumors (see below). Except for their size, the cysts are symptomless unless torsion or accidental rupture occurs. Treatment is by surgical removal.
         b. Serous cystadenomas - These usually do not grow to such huge proportions, are often unilocular, and have a tendency toward papillary excrescences on both their outer and inner surfaces. They are lined by cuboid or short columnar cells, many of which are ciliated. Small calcareous deposits (psammoma bodies) are often seen. These tumors arise from invaginations of the germinal

epithelium of the surface of the ovary. They should be removed surgically.

c. Dermoid cysts - These are cystic teratomas consisting primarily of well-differentiated ectodermal elements (hair, teeth, sebaceous material) but often containing some mesodermal structures also. They probably arise as a parthenogenetic process in the ovarian ova, but other theories of their origin have not been disproved. Diagnosis is often made by the demonstration of teeth in an x-ray film. Torsion of the pedicle of a dermoid cyst is common, since these heavy growths often stretch the pedicle to surprising lengths. The cysts should be removed by shelling out if possible; by oophorectomy if necessary. Since bilateral cysts occur in about 25% of cases, the contralateral ovary should be inspected carefully and bisected if necessary.

2. Malignant -

a. Cystadenocarcinoma - These may be pseudomucinous or serous. There is no way of diagnosing cystadenocarcinoma preoperatively, but since 5-10% of pseudomucinous and 25% of serous cystadenomas are malignant at the time of surgery, all ovarian cysts which are over 5 cm. in diameter or persist for 60 days should be removed. About 10% of all ovarian tumors are cystic carcinomas; about half of these are bilateral. When ovarian malignancy is discovered, panhysterectomy and bilateral salpingo-oophorectomy is indicated. The presence of metastatic implants upon the peritoneum is an unfavorable sign; although irradiation is often of value postoperatively in prolonging the period of regression, cure is not to be expected. The intraperitoneal injection of 1 of the new chemotherapeutic antineoplastic agents may also be of value.

b. Dermoid cysts - Malignant degeneration of a dermoid cyst occurs rarely (see Teratoma, p. 361).

Solid Tumors.

A. Benign:

1. Fibroma of the ovary - An uncommon tumor which may grow to huge size and produces symptoms only by its size or by torsion of its pedicle, except for the rare occurrence of Meigs' syndrome: ovarian fibroma with ascites and hydrothorax (usually right-sided). (It is now known that the ascites and hydrothorax, or hemoperitoneum and hemothorax, may be found in association with any pelvic tumor, benign or malignant.) Treatment is surgical.

2. Brenner tumors - These moderately-sized neoplasms are usually indistinguishable grossly from ovarian fibromas; histologically, they are studded with nests of epithelial cells within dense fibrous tissue, probably originating from Walthard's cell rests. Diagnosis and management are as described for fibroma (above).

3. Other rare tumors - Rarely, such other solid, benign tumors as angioma may be encountered. The symptomatology is not characteristic, and the diagnosis and classification depend

upon surgery and pathologic examination.

B. Malignant:

1. **Carcinoma** - Primary solid carcinoma may take any of several histologic forms which are indistinguishable from each other on gross examination. It has no characteristic color, size, shape, or consistency; and can best be described merely as a solid ovarian tumor which metastasizes and is more likely than a benign tumor to bear evidence of rapid or irregular growth and invasion of its own capsule. It accounts for about 5% of all ovarian tumors; about half are bilateral. Diagnosis and treatment are as described for cystadenocarcinoma (see p. 360).

2. **Sarcoma** - Sarcoma of the ovary is rare. The clinical approach to the problem is as for carcinoma (above).

3. **Teratoma** - Teratomas of the ovary are not common, but must be considered when an ovarian tumor is found in a young woman. Like dermoid cyst, this tumor is probably an example of "imperfect parthenogenesis," consisting of a variety of embryonic tissues which may grow in any conceivable combination of imperfect structures. Metastasis to nearby structures, especially the peritoneum, tends to occur early. One of the tissues in the tumor may grow rapidly, overwhelming the other tissues and thus presenting a tumor which apparently is composed of a single tissue. Struma ovarii, the thyroid tumor of the ovary, is 1 example; pseudomucinous cystadenoma may be a teratoma in which the entoderm has overgrown all other tissues. The treatment of these tumors is by surgical excision, and the prognosis is poor.

4. **Metastatic carcinoma** - Metastasis to the ovary occasionally occurs from the uterus or adjacent pelvic structures or from the gastrointestinal tract (especially the pylorus). It may take the form of a Krukenberg tumor: a firm, smooth, non-adherent, usually bilateral growth which tends to retain the original ovarian contour. This is a mucin-producing tumor, and microscopic examination often shows mucinous cells in which the mucin has flattened the nuclei against the cell wall to produce the typical "signet cells."

Embryonic or Dysontogenetic Tumors.

These are sometimes referred to as functioning tumors or special tumors.

A. **Feminizing, Granulosa—theca Cell Tumors:** (Either or both cell types may be found.) These tumors account for almost 10% of all ovarian malignancies. They may occur at any age; a few have even been reported in prepuberal girls. Since the cells are nearly always functional, the prepuberal patient may demonstrate sexual precocity and the postmenopausal patient may report vaginal bleeding, whereas the woman in the reproductive age group will usually complain of some form of menstrual irregularity - often alternating amenorrhea and heavy bleeding. These tumors must be considered malignant, although the survival rate from granulosa cell tumors is at least 50% and that from theca cell tumors almost 100%. The tumor may recur many years later, and lifetime follow-up is necessary.

The recommended treatment is panhysterectomy and bilateral salpingo-oophorectomy, but more conservative measures may be resorted to in a younger woman who desires future pregnancy and who has an apparently well-confined unilateral tumor. This is a calculated risk of which the patient should be aware. Careful observation is essential.

B. Masculinizing, Arrhenoblastomas (Sertoli—Leydig Cell Tumors): These comprise less than 1% of ovarian neoplasms, and usually occur during the reproductive period of a woman's life. There are no unique gross characteristics. Histologic examination may show a well-differentiated Sertoli cell tubular pattern with interspersed Leydig cells, or there may be an undifferentiated, sarcoma-like appearance. The tumors are not often bilateral, but are malignant in about 20% of the cases reported. They usually exert a masculinizing effect: breast atrophy, enlargement of the clitoris, voice changes, hirsutism, and amenorrhea. The tumor should be removed surgically, and the other pelvic organs should be removed at the same time unless the patient desires future pregnancy and there is good clinical evidence of lack of malignancy. The prognosis is good in most instances.

C. Gynandroblastoma (Mixed Tumor): This rare neoplasm is composed of elements of both granulosa—theca cell tumor and arrhenoblastoma, and may cause any combination of feminizing and masculinizing symptoms.

D. Dysgerminomas (Germinomas, Seminomas): Dysgerminomas are tumors of embryonic germ cells which, because they are still sexually undifferentiated, produce no endocrine-induced changes. (This tumor may also be found in the male, in which case it is called a seminoma.) It is usually malignant and accounts for about 4% of malignant ovarian tumors; about one-third are bilateral. The tumor most commonly occurs between the ages of 10 and 30. The patient may present a picture of retarded sexual development. Any treatment short of panhysterectomy and bilateral salpingo-oophorectomy should be undertaken only with the reasoned consent of the patient.

E. Other Rare Types: There are several rare ovarian tumors (e.g., hypernephromas, adrenal-like tumors, and luteomas) which can only be diagnosed at surgery. Because they are so rare, further discussion in the space available is not feasible.

DISTURBANCES OF MENSTRUATION

Normal menstruation usually starts (menarche) at age 9-17 (avg., 13); it continues (menacme) until cessation (menopause) at about age 45-55 (avg., 47-50). About 50 ml. of blood, mixed with desquamated cells and lacking the ability to clot (fibrinolysins), will be passed in a period of 2-7 days at intervals of 22-35 days.

Variations From Normal Menstruation.

A. Amenorrhea: (Absence of menstruation.)

   1. Primary amenorrhea - (Without previous menstruation.) Primary amenorrhea may be said to exist if the menarche has not occurred by the age of 18; it must be differentiated from cryptomenorrhea, in which endometrium is desquamated from the uterine wall but is retained within the vagina by some (usually congenital) obstruction, e.g., imperforate hymen. Primary amenorrhea may be caused by congenital absence or marked hypoplasia of the uterus and/or ovaries; adrenogenital syndrome; adiposogenital dystrophy (Fröhlich's syndrome); any constitutional deficiency such as anemia or tuberculosis; or marked emotional disturbances.

   2. Secondary amenorrhea - (Following previous menstruation.) With the exception of congenital abnormalities, the causes of secondary amenorrhea are the same as of the primary forms. However, endocrine derangements caused by defeminizing or masculinizing tumors are more apt to occur after the menarche and thus are more apt to be seen in association with secondary than with primary amenorrhea.

B. Hypomenorrhea: (Scanty flow with a normal cycle.) Hypomenorrhea differs from amenorrhea principally in degree. Some women report a scanty flow throughout the menacme. Except for infertility, this condition usually is of no significance, and treatment by extraneous hormone augmentation produces no lasting effect.

C. Menorrhagia: (Excessive flow with a normal cycle.) This disorder is much more common than hypomenorrhea or amenorrhea and is of vastly greater clinical significance. Several common clinical disorders are associated with menorrhagia:

   1 Constitutional factors, such as hypothyroidism or thrombocytopenia or other clotting deficiencies of the blood.

   2. Polyps of the cervix or of the endometrium.

   3. Irregular shedding of the endometrium is a failure of the uterus to desquamate completely within the first 4 days of menstrual flow.

   4. Neoplasms in the pelvis.

   5. Chronic endometritis (rare).

   6. Adenomyosis (see p. 355).

D. Oligomenorrhea: (Infrequent menstruation, i.e., prolonged cycle, but with normal flow.) This may be a precursor of amenorrhea, and any of the pathologic conditions discussed under that heading should be borne in mind. In other instances, oligomenorrhea seems merely to be a normal variation which is of no importance except that it may limit fertility.

E. Polymenorrhea: (Frequent menstruation, i.e., short cycle, but with normal flow.) A cycle of less than 22 days, not commonly seen, is apt to accompany pelvic inflammatory disease or endocrine disturbances. Treatment is by correction of the primary disorder.

F. Metrorrhagia: (Noncyclic bleeding from the uterus.) Metrorrhagia is not caused by endometrial desquamation. The term is thus merely a descriptive one, used to denote a symptom, bleeding.

MISCELLANEOUS CAUSES OF ABNORMAL UTERINE BLEEDING

ANOVULATORY CYCLE

The anovulatory cycle is a failure of ovulation during the men-acme. The cycle is usually prolonged (may be markedly so), the flow may be heavy (see below), and there is almost never any pain even if the woman usually suffers from severe menstrual cramps. Some women have occasional anovulatory episodes with no significant change in cycle or flow.

ENDOMETRIAL HYPERPLASIA

Hyperplasia of the endometrium is proliferation of endometrium without the "ripening" effect of progesterone. It is the result of ex-cessive or (more usually) prolonged exposure to estrogens, whether caused by an anovulatory cycle, an estrogen-producing ovarian tu-mor, or medical administration of estrogens. There is usually a history of amenorrhea followed by profuse bleeding (can be almost exsanguinating). Pain is seldom present.

Curettage controls the bleeding and provides tissue for micro-scopic examination.

If the disorder is caused by an ovarian tumor which can be re-moved, prompt cure is easily obtained. Commonly, however, en-dometrial hyperplasia is caused by anovulatory cycles of unknown etiology, and treatment is more complicated. Repeated curettages may be necessary.

STEIN-LEVENTHAL SYNDROME

The Stein-Leventhal syndrome consists of amenorrhea (or oligomenorrhea) accompanied by obesity and hirsutism in the pres-ence of enlarged polycystic ovaries.

The accepted treatment is surgical removal of a wedge-shaped section of each ovary. About 80% of the patients so treated will re-vert to normal menstrual cycles.

DYSMENORRHEA

Dysmenorrhea (painful menses) may be primary (cause not discernible) or secondary to some other condition (e.g., myoma of the uterus).

Numerous theories of the cause of primary dysmenorrhea have been advanced, but evidence to support them is difficult to obtain. The following syndromes should be ruled out before these explana-tions are resorted to: (1) Pelvic congestion caused by an endocrine-induced fluid (sodium) retention; (2) pelvic ischemia; and (3) endo-crine factors, i.e., over-production or deficiency of estrogen or progesterone.

If the pain of primary dysmenorrhea does not respond to attempts to correct these abnormalities, analgesics should be given. If these are not sufficient to control the discomfort, surgery may be resorted to: hysterectomy if the patient is nearing the menopausal age, or presacral neurectomy for a patient who desires future children. The success rate of neurectomy is about 80-90%.

ENDOMETRIOSIS

Endometriosis is the ectopic occurrence of endometrium. When endometrium invades the myometrium the disorder is termed endometriosis interna or adenomyosis (see p. 355). Endometrium found elsewhere in the body is called endometriosis externa. The ectopic tissue in endometriosis externa is usually in the pelvis (pelvic endometriosis), but it may be found in laparotomy scars, the umbilicus, bladder, the intestinal tract, or almost anywhere in the body. In the pelvis, the most common locations are the ovaries, tubes, the uterosacral ligaments, and the pouch of Douglas, although ectopic endometrial tissue may be seen in any of the genital structures. Endometriosis externa is found in about 15% of women who undergo pelvic operations.

Pathologically, the disease is due to the growth of the ectopic endometrium, usually with intermittent desquamation, under the influence of the ovarian hormones. If growth is in an enclosed space, such as the ovary, repeated episodes of desquamation cause the formation of a cyst containing old blood ("chocolate cyst") which gradually increases in size to a usual maximum diameter of about 10 cm. Surface implants on any tissue cause dense scarring, which draws in the surrounding area and gives a "puckered" appearance which may be white (scarring) or blue (desquamation).

The most common symptom is pain, usually beginning about 1 week before menstruation and reaching its peak with the onset of flow. The site, intensity, and character of the pain depend upon the location and extensiveness of the growth. Implants are commonly in the cul-de-sac, and the pain therefore tends to be low in the midline of the pelvis. Symptoms other than pain may also occur: an implant in the bladder may cause frequency of urination; one in the intestinal tract may cause diarrhea and cramps, etc. Pelvic examination will reveal a mass if the disease has progressed to the stage of cyst formation, but in the earlier stages there may be no palpable abnormalities other than tenderness. An almost pathognomonic finding is tenderness, often with nodularity, of the uterosacral ligaments. This is demonstrated by rectovaginal examination, raising the cervix with the index finger in the vagina to stretch the uterosacral ligaments and facilitate their identification by the middle finger. The clinical diagnosis of endometriosis is usually made on the basis of the history and physical findings; definitive diagnosis depends upon finding the lesion at surgery.

Treatment.

Since the lesion depends upon ovarian hormones for its growth, castration by surgery, irradiation, or spontaneous menopause will cause regression. However, this is often undesirable, as when the patient wishes to avoid sterility.

A. Medical Treatment: Medical management will not cure but usually should be tried first as a palliative measure unless a mass is present. For practical purposes, analgesics and treatment of pelvic congestion are the most dependable medical measures. Large doses of stilbestrol may give temporary relief by halting the ovarian cycle. Treatment with recently developed hormones may prove to be beneficial.

   The most effective nonsurgical "treatment" is pregnancy, which halts the process for almost a year.

B. Surgical Treatment: Hysterectomy and bilateral salpingo-oophorectomy is the surgical procedure of choice if the disease is widespread or if the patient is nearing the menopause and has no desire for future pregnancies. Irradiation castration is not recommended except in the most unusual circumstances. Conservative treatment is indicated for the younger woman who wishes future pregnancies, leaving intact all or part of 1 ovary, a tube, and the uterus if possible. When operating upon a patient with endometriosis who desires future pregnancies, the surgeon's task is to attempt to preserve and repair tissues, not merely to remove them. This "conservative" type of operation may be inadequate, and further surgery may be necessary at a later date; but if the patient has a complete understanding of the situation it is often worth trying.

   In the patient who has had total ablation of the ovaries but who still has endometriosis, exogenous estrogens given for the purpose of controlling the menopausal syndrome will rarely cause recrudescence of the implants.

RETRODISPLACECENT OF THE UTERUS

The normal position of the uterus is considered to be anteflexion but in about one-third of women it will be either retroverted (turned backward on its transverse axis) or retroflexed (bent backward).

The retrodisplaced uterus does not commonly cause any symptoms. Occasionally, however, retrodisplacement causes low backache, dysmenorrhea, menorrhagia, and/or dyspareunia.

A pessary will bring relief. The pessary may be worn intermittently - 1-2 months at a time, with a period between for the purpose of sparing the vaginal mucosa - until the menopause, after which time the malformation rarely causes discomfort. In rare instances it may be advisable to suspend the uterus surgically, but the failure rate of this procedure is high.

PELVIC RELAXATION

Relaxation of the tissues of the female pelvis occurs to some degree in most women, although it is often asymptomatic. Weakening of the tissues may be due to congenitally poor tissue tone, neurogenic deficiency (e.g., occult anterior spina bifida), and/or childbirth lacerations. Relaxation of tissues in specific areas causes specific disturbances:

Prolapse of the uterus, caused by a relaxation of the uterine supports (especially the cardinal and uterosacral ligaments), may be first degree (early), second degree (cervix to the introitus), or third degree (cervix past the introitus). The only symptom is a "dragging sensation" in the pelvis.

Urethrocele, a sagging of the urethra caused by relaxation of the lower portion of the pubocervical fascia, produces stress incontinence and urgency of urination.

Cystocele, a bulging or prolapse of the bladder caused by weakening of the entire pubocervical fascia, encourages cystitis, frequency, and urgency of urination.

Rectocele, an anteriorly-directed bulging of the rectum due to weakness of the fascial tissues overlying the rectum produces constipation which often may be relieved by finger pressure in the vagina.

Enterocele, a true hernia of the peritoneum of the pouch of Douglas, extending caudad into the rectovaginal septum, may produce pelvic heaviness but often causes no symptoms.

Perineal relaxation is a lack of firm tissues at the introitus caused by a weakening of all fascial planes and muscles of the area. Although this condition may be due to nonobstetric causes, childbirth lacerations are the most common factor. The tearing of the tissues may have been subcutaneous, in which case scars will not be apparent upon inspection, but the more common finding is a perineal laceration which has left its telltale scar. Symptoms are pelvic heaviness and dragging.

Treatment.

Surgical repair by any of several technics (e.g., the Manchester operation, with or without vaginal hysterectomy) is usually the treatment of choice, but the failure rate is high since relaxation of pelvic tissues is commonly seen in women with an inborn tendency toward poor tissues. Even in the best hands, about 10% of operative results are less than satisfactory no matter what technic is used.

A pessary may be used if the patient is unable to withstand surgery. Special exercises of all the perineal muscles may be beneficial, but patients rarely perform them faithfully.

FISTULAS

The most commonly seen fistulas involving the female genital tract are rectovaginal, vesicovaginal, and urethrovaginal fistulas. They should be watched for in association with malignancy or following pelvic surgery or radiotherapy, and are occasionally seen following trauma (e.g., difficult delivery).

The most common complaint is incontinence of urine, feces, or intestinal gas; other symptoms are not commonly present. The lesion can usually be seen by vaginal inspection, but the aperture may be so small or so well concealed in folds of mucosa that it is not easily discovered. In these instances the suspected origin of the tract can be filled with a methylene blue solution and the vagina inspected for emergence of the dye.

Small, recent fistulas may close spontaneously, especially if healing is encouraged by a freshening of the wound edges, silver nitrate, and diversion of the urinary or fecal stream by catheter or tube. Usually, however, the only effective treatment is surgical repair. This may be very difficult, and the failure rate is high.

OBSTETRIC PROBLEMS

ABORTION

Abortion is defined as termination of pregnancy before the fetus becomes viable (about 28 weeks), it occurs in about 10-15% of all pregnancies, usually in the second or third month. Habitual abortion is said to exist after 3 successive spontaneous abortions.

Classification and Treatment.
A. Spontaneous Abortion.
1 Threatened abortion - Bleeding and cramps, with the cervix dilated 1 cm. or less. Treatment consists of bed rest, sedation, and analgesics.
2. Inevitable abortion - The cervix is now dilated to 1 cm. or more, or the membranes rupture. A conservative course may be followed and complete abortion awaited if bleeding is not too severe. Otherwise, dilatation and curettage are indicated.
3. Incomplete abortion - Part of the products of conception (usually the fetus) has been expelled, but part (usually placental fragments) remains behind. Treatment consists of dilatation and curettage.
4. Complete abortion - The entire contents of the uterus have been expelled. Since nothing remains behind but decidua basalis and parietalis, which will spontaneously desquamate, no treatment is indicated. If excessive bleeding and/or cramps occur after a diagnosis of complete abortion has been made, then the diagnosis was in error and dilatation and curettage are indicated to remove retained fragments.
B. Induced Abortion:
1. Therapeutic - Indicated for reasons of maternal health or inevitable fetal malformation.
2. Criminal - Dilatation and curettage should be performed if abortion is incomplete.
C. Septic Abortion: Septic abortion is abortion at any stage or of any type complicated by infection.
D. Missed Abortion: Missed abortion is intrauterine retention of a dead embryo for 2 months or more. Dilatation and curettage are indicated if spontaneous abortion does not occur after estrogen-induced withdrawal bleeding.
E. Habitual Abortion: Three consecutive abortions.

Prognosis for Future Pregnancies.
An uncomplicated spontaneous abortion, when due to a blighted ovum (rather than to an abnormality of the maternal structures), does not compromise future pregnancy.

HYDATIDIFORM MOLE

Hydatidiform (vesicular) mole is a benign proliferation, probably neoplastic, of the trophoblast which produces cystic degeneration of the villi and, usually, fetal death. It occurs once in about 2000 pregnancies. Grossly, the hydropic villi appear as vesicles a few mm. to about 2 cm. in diameter.

The presence of a mole might be suspected if the uterus does not grow at the normal rate, if there is bleeding, if lutein ovarian cysts (from stimulation from the hyperplastic trophoblast) are present, if the fetal skeleton is absent, or if a high titer of chorionic gonadotropin is present (demonstrated by a positive biologic pregnancy test on an unusually small amount of test material). Hydatidiform mole should be removed immediately. If the mole is larger than a three-month pregnancy, abdominal hysterotomy may be preferable. After evacuation, the patient should be observed closely for 2 years with repeated biologic pregnancy tests: a positive test in the absence of pregnancy indicates malignant degeneration (see Chorio-epithelioma, below).

CHORIO-EPITHELIOMA

Chorio-epithelioma or choriocarcinoma, a rare condition, is a malignancy of fetal trophoblast which may be seen following term delivery or abortion but is usually a complication of hydatidiform mole. Although over 50% of cases of chorio-epithelioma result from moles, only 1-2% of moles lead to chorio-epithelioma. This neoplasm is extremely malignant; early invasion and metastasis to distant sites (especially the lungs) is the rule.

Diagnosis is made during the two-year follow-up care recommended for all cases of hydatidiform mole. The patient is watched for new growths in the vaginal mucosa, abnormal bleeding, or persistently positive pregnancy tests. If chorio-epithelioma is found in the uterus, immediate hysterectomy and bilateral salpingo-oöphorectomy is indicated plus the surgical removal of any metastatic lesion which can be completely removed. Postoperative irradiation is usually considered advisable, but statistical proof of its value is lacking. Amethopterin (Methotrexate®), a new chemotherapeutic agent, is giving hopeful results in some cases.

The prognosis is poor. Most patients survive less than 1 year after diagnosis. However, this malignancy is so unpredictable that strenuous efforts at control should always be made even in apparently hopeless situations.

ECTOPIC PREGNANCY

An ectopic pregnancy is one which follows implantation of an ovum on any surface other than the uterine cavity, usually in the tube but occasionally in the ovary, the cervix, or the peritoneum.

TUBAL PREGNANCY

Tubal gestation occurs about once in 250 pregnancies, often secondary to a congenital or inflammatory tubal disorder.

Clinical Findings.
A. Symptoms and Signs: Pain is usually unilateral at first. It increases in severity and becomes generalized with any leakage into the peritoneal cavity. Pain becomes acute when the tube ruptures. Vaginal bleeding is irregular in amount and does not occur at the expected time of menstruation. Shock will be present if the tube ruptures.

The mass in the pelvis is usually quite tender (sometimes excruciatingly so), may or may not be fixed, and tends to grow. The uterus itself is usually soft and often somewhat enlarged.

After implantation of the ovum, the course of the condition depends upon the effects produced by the invading trophoblast, as follows:

1. In most instances the growing ovum stretches the tube to the point where it suddenly ruptures, causing extensive internal bleeding. This produces severe pain, shock, and the clinical picture of an acute abdomen due to internal hemorrhage.

2. The ovum may die soon after implantation; the patient's pain, which may have been of any degree of severity, will then gradually subside and there will be no further ill effects.

3. If the ovum was implanted near the end of the tube, it may grow to 1-3 cm. in diameter and then be extruded out of the fimbriated end of the tube into the cul-de-sac ("tubal abortion"). When this happens the character of the pain will change: unilateral discomfort will suddenly become acute, spread to both lower quadrants and the rectum, and then will gradually subside over a period of about 1 month. Internal bleeding is rarely severe, and surgery is seldom indicated.

4. The invading trophoblast may reach the serosa of the tube without having ruptured a vessel of significant size; internal bleeding usually develops so slowly that the abdominal crisis is avoided. In this instance the pain is usually constant and more or less disabling, but operative intervention is often delayed because there is no crisis and no definite diagnosis. The pain usually persists until the disorder is corrected at surgery.

5. The growing trophoblast may invade through the tube wall without causing sufficient pain to bring the patient to surgery, and may then grow along the tube, broad ligament, and often the adjacent structures (uterus, pelvic wall, intestine) as an abdominal pregnancy (see p. 372).

B. Laboratory Findings: A pregnancy test may be either positive (trophoblast functioning) or negative (trophoblast not functioning); endometrial biopsy or curettage may or may not demonstrate decidual endometrium for the same reason.

C. Special Examinations: Free blood in the peritoneal cavity can be demonstrated by needle aspiration through the cul-de-sac. Culdoscopy is useful in the diagnosis of ectopic pregnancy in selected cases; however, since a previous pelvic infection so

often is the cause of a tubal pregnancy, the tube is apt to be adherent to the peritoneum of the cul-de-sac, which renders culdoscopy of no value.

Differential Diagnosis.

Tubal pregnancy must be differentiated from pelvic inflammatory disease, ovarian cyst, and threatened or incomplete abortion of an intrauterine pregnancy.

Treatment.

Treatment consists of surgical excision of the mass and reconstruction of the tube if technically feasible, or removal of the entire tube if necessary. If it seems certain that tubal abortion or spontaneous death of the embryo in the tube has occurred, observation may be indicated, but the patient should be in the hospital and the physician should be prepared for surgery at any time. When there has been a tubal rupture, bleeding may be massive; transfusion and antishock measures are important adjuncts to the surgical procedure.

OTHER FORMS OF ECTOPIC PREGNANCY

Interstitial Pregnancy.

Interstitial pregnancy is a pregnancy in that portion of the fallopian tube which lies in the wall of the uterus. The unilateral enlargement may be discovered by pelvic examination if the patient is not too obese.

Because of the thick myometrium, rupture may not occur until the embryo has grown for several months. Rupture is a serious matter since the uterine and ovarian arteries are often involved. Surgical excision of the cornua of the uterus before rupture can occur is advisable.

Cornual Pregnancy.

A cornual pregnancy is one in which the ovum was implanted in the uterine horn. If severe symptoms or rupture occurs, interstitial pregnancy is the more likely diagnosis. Cornual pregnancy often causes sacculation of that portion of the uterus, with consequent adjustment and term pregnancy.

Cervical Pregnancy.

Implantation of the ovum in the cervical mucosa usually precipitates pain and bleeding early in pregnancy. The bleeding may assume critical proportions if the pregnancy progresses past 3 months. If evacuation through the vagina and packing do not control bleeding, hysterectomy is indicated.

A similar clinical picture may be presented by the imprisonment of a spontaneously aborted ovum in the cervical canal because of a stricture of the external os of the cervix (cervical abortion). Dilatation of the external os will permit the delivery of the abortus.

Ovarian Pregnancy.

Ovarian pregnancy presents a clinical picture identical with that of pregnancy of the fimbriated end of the tube, and treatment is the same, i.e., surgical removal.

Abdominal Pregnancy.

Abdominal pregnancy may occur as a primary nidation or, secondarily, when a tubal pregnancy grows through the wall of the tube and extends over the peritoneal surface to adjacent structures. It may go to term, although the course of the pregnancy usually includes episodes of pain and bleeding. Suspicion should be aroused by prolonged pregnancy, failure of the cervix to efface and begin dilatation, and abdominal palpation of fetal small parts.

Treatment is by laparotomy. If the blood supply to the placenta cannot be completely controlled by ligature, the placenta should be left in situ, attached wherever it is situated. If spontaneous absorption does not occur, the placenta will wall off and liquefy, and can be drained later.

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Urology

GENERAL PRINCIPLES OF DIAGNOSIS

The essential steps in the workup of the urologic patient are
(1) a history and physical examination, (2) a routine laboratory
evaluation, and (3) either a plain film of the abdomen or excretory
urograms. The laboratory tests should include a stained smear of
the urinary sediment and a PSP test.

In certain instances more definitive investigative procedures
may be necessary, e.g., passage of urethral catheters or sounds,
cystometry, special radiographic technics, cystoscopy, ureteral
catheterization, and retrograde urograms.

UROLOGIC LABORATORY EXAMINATION

Urethral Discharge.
Urethral discharge should be examined immediately in saline
for trichomonads. It should then be stained and examined micro-
scopically for bacteria.

Urinalysis.
- A. Collection of the Specimen: In the male a "midstream"
  specimen must be obtained. In the female a similar "mid-
  stream" or catheterized specimen can be collected.
- B. Immediate Examination: Immediate examination is imperative.
  A normal urine specimen contains not more than 1-2 red or
  white cells/low power field. Whether pus cells are present
  or not, the sediment must be stained with methylene blue since
  chronic urinary tract infections are apt to be apyuric (and
  asymptomatic). If pus cells are present but no bacteria can be
  found in the stained smear, acid-fast stains are indicated.

  The presence of bacteria on a stained smear implies that
  there are at least 10,000 organisms/ml. of urine; this is '
  pathognomonic of clinical infection.
- C. Cultures: Only quantitative cultures should be ordered; quali-
  tative cultures are too often falsely positive. More than 1000
  colonies/ml. makes the diagnosis of urinary infection. Sensi-
  tivity tests may be indicated. Cultures for tubercle bacilli
  should be utilized in patients suspected of having urinary
  tuberculosis.
- D. Sulkowitch's Test: This test affords a rough estimate of the
  amount of calcium in the urine. To 5 ml. of urine add 2 ml.
  of Sulkowitch's reagent. A slight cloud implies a small amount
  of calcium; the rapid formation of a very dense cloud means
  hypercalcemia until proved otherwise.

Renal Function Tests.

- A. **Morning Specific Gravity:** If this is 1.024 or higher in the absence of massive proteinuria, total renal function is good.
- B. **The PSP (Phenol Red) Test:** This test accurately measures total renal function and makes possible an estimation of the amount of residual urine. Do not force fluids before or during the test. The patient should void just before the injection of 1 ml. of the dye.

    Regardless of the volume of urine, the normal amount of PSP excreted in one-half hour is 50-60%. Recovery of less than this amount means either impaired renal function or vesical or bilateral ureteral obstruction. In this instance, a second half-hour specimen should be collected. If there is no residual urine, the second specimen should contain 10-15% of the dye. If more than this is recovered, residual urine is present; the amount of residual urine can then be estimated as shown by the following example:

    > First half-hour = 35 ml. = 30%
    > Second half-hour = 25 ml. = 25%

    The curve is "flat," yet the one-hour total is good; renal function is therefore normal and residual urine is present. Since the first specimen should have contained 50-60% of the dye, the residual urine is 30-35 ml.

- C. **Tests** for nitrogen retention should be performed if the PSP excretion is less than 30% in one-half hour.

Radiography.

- A. **Noninstrumental:**
    1. **Plain film of the abdomen (KUB)** - This may show evidence of a ruptured viscus, gallstones, bowel obstruction, differences in size or position of the kidneys, calcifications, and diseases of bone (e. g., metastatic carcinoma).
    2. **Excretory urograms** - These films will establish the diagnosis of most diseases of the kidneys and ureters.
    3. **Retroperitoneal pneumograms** - Oxygen introduced into the presacral space will usually reveal the kidneys and adrenals in sharp contrast. Tomography is essential to the diagnosis.
    4. **Gastrointestinal studies** - These are useful in differential diagnosis. Retroperitoneal masses (e. g., enlarged kidney) may displace intraperitoneal organs.
    5. **Aortograms** - Aortograms will demonstrate renal tumors or cysts and stenosis of the renal arteries.
- B. **Instrumental:**
    1. **Urethrograms** - A viscous radiopaque solution instilled into the urethra will reveal the caliber of the urethra and enlargement of the prostate.
    2. **Cystograms** - The instillation of radiopaque material into the bladder will demonstrate rupture of that organ, vesical diverticula, and ureteral reflux.
    3. **Retrograde urograms** - If excretory urograms are not diagnostic or if maximum detail is essential, catheters may be passed to the renal pelves through the cystoscope. Concentrated radiopaque solution can then be introduced into the

renal pelves. In addition, bacteriologic studies can be made of the urine specimens from each kidney and separate renal function tests can be done.

Instrumental Examination of the Urinary Tract.
  A. Catheter: The urethral catheter is useful for exploration of the urethra and the measurement of residual urine.
  B. Sounds: Metal sounds may be used to diagnose urethral stricture.
  C. Cystoscope: This instrument permits inspection of the urethra and bladder as well as manipulation of ureteral stones, biopsy of vesical tumors, and catheterization of the ureters.

URINARY OBSTRUCTION AND STASIS

Etiology.
  Congenital anomalies, more common in the urinary tract than in any other organ system, are the most frequent causes of obstruction. Other congenital causes of stasis of the urine are spina bifida or myelomeningocele, which cause nerve damage, particularly to the sacral roots. In adult life many acquired obstructions occur. The common causes are stricture secondary to infection or injury, prostatic disease, vesical tumor or local extension of cancer, and stone.

Clinical Findings.
  A. Symptoms and Signs:
    1. Lower and mid tract (urethra and bladder) - The principal symptoms are hesitancy, lessened size and force of the stream, terminal dribbling, and urinary retention. Rectal examination may show atony of the sphincter (which suggests interruption of the sacral roots) and enlargement or cancer of the prostate. Evidence of distention of the bladder may be found on suprapubic percussion.
    2. Upper tract (ureter and kidney) - The principal complaints are pain in the flank, hematuria (from stone), and gastro-intestinal symptoms. If infection ensues there may be chills, fever, and vesical irritability. If advanced bilateral hydronephrosis is present, the only symptoms may be those of uremia. An enlarged kidney may be found. Cancer of the cervix may invade the bladder, thus occluding 1 or both ureteral orifices. A large pelvic mass (e.g., a gravid uterus) can compress the ureters.
  B. Laboratory Findings: Anemia may be present, secondary to chronic renal infection or uremia. Pus and bacteria may be present in the urine. If unilateral hydronephrosis is present, the PSP excretion will be normal. Depression of PSP excretion implies bilateral renal damage or vesical or bilateral ureterorenal residual urine. If bilateral renal damage is severe, nitrogen retention will be demonstrated.
  C. X-ray Findings: A plain film of the abdomen may show enlargement of the renal shadows, calcific bodies suggesting urinary stones, or metastases to bone. Excretory urograms

will establish the diagnosis unless renal function is so poor that the radiopaque fluid is not excreted. They will demonstrate dilatation of the upper tract and will localize calcific bodies in or outside of the urinary tract. The accompanying cystogram may reveal trabeculation and diverticula if the obstruction is distal to the bladder. Vesical tumors, nonopaque stones, and large intravesical prostatic lobes may cast radiolucent shadows. A film taken immediately after voiding may show residual urine.

Retrograde cystography (see p. 374) may show changes in the bladder wall caused by distal obstruction. If the ureterovesical valves are incompetent, excellent ureteropyelograms will be obtained because of ureteral reflux. A film taken during the act of voiding may also demonstrate reflux.

Retrograde urograms may be necessary to more clearly delineate the site and type of ureteral obstruction.

D. Instrumental Examination: Passage of a catheter will be arrested by a stricture. Catheterization may reveal residual urine. Cystoscopy will permit visualization of a urethral stricture, enlarged prostate, obstructing vesical tumors, and changes secondary to distal obstruction (e.g., trabeculation). Catheters may be passed to the renal pelves for estimation of function of each kidney, the presence of renal infection, and retrograde pyelograms.

Differential Diagnosis.

A mass in one or both flanks suggests polycystic disease, tumor, or hydronephrosis. Such a finding requires radiologic study.

Complications.

Infection is the principal complication of stasis.

Treatment.

A. Relief of Obstruction:

1. Lower tract obstruction - Urethral strictures must be dilated or corrected surgically. Bladder neck (prostatic) obstruction must be treated. If ureterovesical reflux is present, constant drainage of the bladder by indwelling catheter or cystostomy must be maintained. If reflux persists for many months, surgical repair of the ureterovesical valve may be necessary.

2. Upper-tract obstruction - If tortuous, kinked, dilated or atonic ureters have developed secondary to lower tract obstruction (so that they are themselves obstructive), vesical drainage may not protect the kidneys from damage. Nephrostomy may then restore ureteral function, thus allowing removal of the nephrostomy tube.

   The relief of obvious ureteral obstruction (e.g., ureteral stone) is mandatory. If tests of renal function and urography show that 1 kidney is badly damaged, nephrectomy may be necessary.

B. Eradication of Infection: Every effort must be made to combat secondary infection. If infection has been severe and prolonged, antibiotic therapy may be unsuccessful (see p. 383).

Prognosis.

If renal function is fair to good, if the obstruction can be corrected, and if infection can be eradicated, the prognosis is generally excellent.

INFECTIONS OF THE GENITOURINARY TRACT

The "nonspecific" infections are a group of diseases having similar manifestations and caused by gram-negative rods (e.g., Escherichia coli, Proteus vulgaris) or, less commonly, gram-positive cocci (e.g., Streptococcus faecalis, Staphylococcus aureus). The "specific" infections are caused by specific bacteria (e.g., Mycobacterium tuberculosis, Neisseria gonorrhoeae), each of which causes a clinically unique disease.

The factors contributing to infection are (1) the presence of obstruction and stasis, (2) the presence of a foreign body (e.g., renal stone), and (3) lowered body resistance.

ACUTE PYELONEPHRITIS

Acute pyelonephritis is manifested by severe aching over the involved kidney and symptoms of cystitis (see p. 379). The temperature reaches 102-104°F. (38.9-40°C.), often with chills. Nausea and vomiting are usually present. Tenderness over the kidney, muscle spasm, abdominal distention, and rebound tenderness may be noted. The WBC is elevated and the percentage of neutrophils increased. Urinalysis reveals white blood cells and bacteria. Quantitative cultures are positive (see p. 373). Sensitivity tests may be helpful in refractory cases. Renal function tests are normal (see p. 374).

Excretory urograms are normal. Their value lies in demonstrating the presence of obstruction (see p. 375) as the cause of the infection.

Urinalysis and urography differentiate pyelonephritis from pancreatitis, appendicitis, and gallbladder disease.

Treatment.
- A. Specific Measures: A sulfonamide or one of the tetracyclines is indicated (see p. 614). If the patient does not respond to treatment in 48 hours, either the wrong drug is being used or obstruction is present. Change to a more promising drug and take excretory urograms.
- B. Follow-up Care: Even after clinical response, the urinary sediment must be examined for pathogens for at least 2 months.

Prognosis.

The prognosis is good if response to antibiotics is complete. If obstruction is present but undiagnosed, recurrences are to be expected. Uncontrolled infection may become chronic.

CHRONIC PYELONEPHRITIS

Chronic pyelonephritis is a common disease which often goes undiagnosed. Unsuspected chronic pyelonephritis is found in 10-15% of autopsies. Diabetics are particularly susceptible.

Clinical Findings.
  A. Symptoms and Signs: There are apt to be few if any symptoms except at the time of exacerbation, when the patient may complain of fever, back pain, and vesical irritability. If the disease is advanced and bilateral, the presenting symptoms may be those of uremia. There are usually no physical findings.
  B. Laboratory Findings: The WBC may be elevated during acute exacerbations. The urinary sediment often contains few if any white cells, but some bacteria can always be found on the stained smear. Quantitative cultures and sensitivity tests must be performed.
     The PSP or other renal function tests will be normal in unilateral renal infection. If total renal function is depressed, bilateral damage or urinary stasis must be assumed to be present.
  C. X-ray Findings: Excretory urograms may be normal in the early stages. As parenchymal scarring progresses, the infundibula narrow and obstruct the calyces, which become dilated (clubbed); decreasing function causes delayed appearance and lack of concentration of the radiopaque substance.
  D. Instrumental Examination: The bladder may show changes due to chronic infection. The introduction of ureteral catheters will identify the site of infection and permit retrograde urography. The function of each kidney can be measured by the PSP test.

Differential Diagnosis.
  Urinalysis and urography differentiate chronic pyelonephritis from recurrent acute cystitis, chronic cystitis, and tuberculosis.

Complications.
  Hypertension and stone formation are the principal complications. Uremia may supervene if the disease is bilateral.

Treatment.
  A. Specific Measures:
     1. Medical - Intensive antimicrobial therapy is needed (see p. 614). The choice of drug depends upon sensitivity tests. The drug should be given for 2-4 weeks or longer and followed by "suppressive" therapy with small daily divided doses of 1 of the sulfonamides, nitrofurantoin, or mandelamine mandelate given for months. In a significant number of cases chronic renal infection cannot be eradicated; suppressive therapy with small doses of chemotherapeutic drugs is then necessary for months or years.
     2. Surgical - Correction of obstruction (e. g., ureteral stenosis) may be indicated. It may be necessary to remove a badly damaged kidney.
  B. Treatment of Complications: When renal function is impaired

bilaterally, a urine output of at least 1500 ml. is necessary to facilitate the removal of metabolic waste products.

Prognosis.

Prognosis should be guarded, for medical treatment often fails to cure the infection. Fortunately, the usual etiologic bacteria do not rapidly destroy renal tissue; longevity may not therefore be jeopardized.

ACUTE CYSTITIS

In the female, cystitis is commonly caused by ascent of bacteria up the urethra. In men, cystitis is never primary but occurs only as a complication of prostatitis, pyelonephritis, or vesical residual urine (e.g., enlarged prostate). Infections of the bowel may involve the bladder by contiguity.

Symptoms and signs include urethral burning on urination, urgency, frequency, and, often, terminal hematuria. Fever is low-grade or absent unless prostatic or renal infection is present. A history of recurring attacks suggests chronic cystitis or pyelonephritis, prostatitis, or urinary stasis. Rectal examination may reveal an atonic anal sphincter (neurogenic bladder) or an enlarged or cancerous prostate. The gland should not be massaged during the acute phase of the vesical infection. Urinalysis shows pus and bacteria, red cells are often present. Renal function is normal.

Urinalysis and response to treatment differentiate acute cystitis from chronic prostatitis, chronic cystitis, and tuberculosis. Cystoscopy will reveal vesical neoplasm.

Acute pyelonephritis may develop.

The sulfonamides are the most useful drugs for the treatment of acute cystitis (see p. 614). If they fail to sterilize the urine in 14 days, a thorough urologic investigation is indicated.

In the absence of stasis, acute cystitis resolves promptly with medical therapy. If infection recurs, the underlying cause must be determined.

CHRONIC CYSTITIS

Chronic cystitis is often secondary to chronic renal infection or residual urine. Complaints may be those of constant or recurring mild vesical irritability, or there may be none at all.

Few or no pus cells may be found in the urine; nevertheless, the stained smear will show bacteria. Cultures will be positive. Renal function tests are normal. Excretory urograms are normal in uncomplicated cystitis. The post-voiding film may reveal residual urine. The attempted passage of a large catheter (22 F.) may reveal urethral stricture or residual urine. Cystoscopy may show evidence of cystocele, vesical stone, or bladder neck obstruction.

Urinalysis, urography, and response to therapy differentiate this disease from chronic pyelonephritis and tuberculosis.

The sulfonamides (see p. 614) may be tried first, but they often fail to cure longstanding infections. If infection still persists, cultures and sensitivity tests should be obtained. Antibiotic

treatment must be intensive and prolonged (3-4 weeks). Simple drug therapy often fails to eradicate the infection unless steps are taken to treat the cause.

ACUTE PROSTATITIS AND PROSTATIC ABSCESS

Acute prostatic infection is usually hematogenous in origin. It usually resolves with or without treatment, but in rare cases progresses to abscess formation. Vesical irritability is extreme. High fever is to be expected. Urinary retention may occur. Rectal examination may reveal an enlarged, hot, tender prostate; massage is contraindicated at this time.

Leukocytosis is present. Urinalysis shows white and red blood cells and bacteria.

Rectal examination differentiates from acute pyelonephritis and prostatic enlargement. If an abscess forms, it may rupture into the urethra or perineum. Surgical drainage may be necessary.

The tetracyclines and chloramphenicol (see p. 614) are the drugs of choice. Response is usually prompt. A few weeks later, the residual infection must be treated (see Chronic Prostatitis, below).

The prognosis is good. If antibiotic treatment is inadequate, the infection may become chronic.

CHRONIC PROSTATITIS

Prostatic infection often has a hematogenous source, but bacteria can also ascend the urethra or descend from the bladder or kidney. An acute prostatitis can become chronic.

There are usually no symptoms. Symptomatic prostatitis may cause urethral discharge or vesical irritability from secondary cystitis. The presenting complaint may be acute epididymitis (see p. 381). The presence of epididymitis implies prostatitis. The prostate may feel normal or may have areas of induration. Prostatic massage will produce a secretion containing pus cells. The urine may be infected. Excretory urograms may show prostatic calculi or vesical residual urine.

Cystitis in men is always secondary to renal or prostatic infection or residual urine. A PSP test and examination of the urine and prostatic secretion will make the differentiation. Complications include urethritis, cystitis, and epididymitis.

Response to chemotherapeutic agents (see p. 614) is not spectacular, but they should be employed. Secondary cystitis usually responds rapidly.

Prostatic massage performed at intervals of 10-14 days promotes drainage. Intercourse should be encouraged for the same reason.

Chronic prostatitis in itself causes little harm, but its complications do. For this reason, routine prostatic massage should be done in all men so that the infection can be discovered and treated.

URETHRITIS IN THE MALE

Acute urethritis may be caused by nonspecific bacteria, trichomonads, or gonococci. The symptoms are urethral discharge and, at times, burning on urination. The infection may be secondary to prostatitis.

The discharge should be examined in saline for trichomonads and stained for nonspecific organisms and gonococci. The prostatic secretion should be examined for trichomonads and pus; if either is present, a series of prostatic massages should be given at intervals of 10-14 days.

Nonspecific urethritis usually responds to antibiotics given for 1 week (see p. 614). Gonorrhea seldom fails to clear when 3 daily injections of 600,000 units of penicillin are given.

Chronic urethritis presents with mild urethral discharge. Bacteriologic study, as noted above, should be done. It usually responds to the antimicrobial drugs (see p. 614). In resistant cases, including trichomoniasis, daily instillations of 8 ml. of 0.25% strong silver proteinate (Protargol®) solution are usually curative. If trichomonads are found, the sexual partner should also be treated; a condom should be used.

ACUTE EPIDIDYMITIS

Acute nonspecific epididymitis is secondary to prostatitis and is a common complication of prostatectomy unless prophylactic vasoligation is performed. The fibrosis which occurs with healing may obstruct the ducts. If bilateral, sterility may ensue.

The symptoms include marked pain and swelling of the epididymis which soon involves the testicle. Fever may be as high as 102-104°F. (38.9-40°C.). Symptoms due to secondary cystitis may be present (see p. 379).

Only in the early stages can the enlarged, tender epididymis be felt separate from the testis. The scrotal skin is often reddened and adherent to the inflammatory mass. Prostatic massage is contraindicated in the acute stage, since it may make the epididymitis worse. Urinalysis may show infection.

Acute epididymitis must be differentiated from tuberculosis, testicular tumor, torsion of the spermatic cord, and mumps orchitis

Treatment consists of cold compresses, antimicrobial drugs (see p. 614), and bed rest with support to the scrotum. Analgesics (e.g., codeine; see p. 605) are required for pain.

ACUTE ORCHITIS

Acute orchitis most commonly occurs as a complication of mumps. It is most often unilateral.

The symptoms and signs include painful swelling of the testicle, fever, and parotitis. Except for leukocytosis, the laboratory evaluation is normal.

Acute orchitis must be differentiated from acute epididymitis (see above) and torsion of the spermatic cord (see p. 404).

The main complication is atrophy and, if bilateral, infertility.

Androgenic function, however, is maintained.

Infiltration of the spermatic cord with 1% procaine may abort the disease. Bed rest, analgesics, cold compresses, and scrotal support are also helpful.

GENITOURINARY TUBERCULOSIS

Genitourinary tuberculosis is a disease of young adults. The infecting organism is Mycobacterium tuberculosis, which reaches the genitourinary organs by the hematogenous route. The kidney is most often affected. Secondary involvement of the ureter, bladder, prostate, and epididymis is common.

The renal lesion is typified by caseation and ulceration of the mucosa of the calyces. Calcification is often present.

In the early stages, the bladder merely shows the nonspecific change of hyperemia. Later, tubercles appear in the mucosa; they may coalesce and ulcerate. In the healing process, fibrosis may be marked; ureteral occlusion may result.

The infection often descends to the prostate, which becomes nodular secondary to the fibrosis caused by healing; the seminal vesicles likewise may be indurated. Similar changes may develop in the vas and epididymis. Rarely, the epididymal infection may involve the testis by direct extension.

Clinical Findings.
A. Symptoms and Signs: The renal lesion is usually silent. Symptoms of cystitis are the common complaint. Only occasionally is tuberculous epididymitis painful.

Evidence of extra-urologic tuberculosis may be noted. A thickened, nontender epididymis may be found, and is often associated with a nodular vas deferens. A draining scrotal sinus may be present. Rectal examination may reveal induration and nodularity of the prostate and seminal vesicles.

B. Laboratory Findings: Pus without organisms on a methylene blue stain of the urinary sediment means that tuberculosis is present until proved otherwise. Acid-fast stains will reveal tubercle bacilli in 60% of cases. Cultures for tubercle bacilli and guinea pig inoculation are positive in almost every case of urinary tuberculosis.

Renal function is normal unless the infection is bilateral and severe. A negative tuberculin test in an adult speaks against the diagnosis.

C. X-ray Findings: A chest film may show active or healed tuberculous lesions. A KUB may reveal punctate calcification in the renal parenchyma. Excretory or retrograde urograms may show "motheaten" (ulcerated) calyces, obliteration of calyces, or a straight ureter due to contracture from scarring.

D. Instrumental Examination: Cystoscopy may reveal the typical tubercles; biopsy may establish the diagnosis. Ureteral catheterization will afford urine specimens for bacteriologic study.

Differential Diagnosis.
Acid-fast stains and excretory urography differentiate tuberculosis from chronic nonspecific cystitis or pyelonephritis.

Nonspecific epididymitis is very painful at onset; tuberculous epididymitis may be but is usually not painful. The finding of pyuria with tubercle bacilli is diagnostic. Excretory urograms may show the typical renal lesion.

Complications.
If vesical involvement is severe, secondary fibrosis may cause ureteral occlusion with hydronephrosis. Bilateral epididymitis will cause infertility. The testis may be destroyed by direct extension.

Treatment.
Tuberculosis of the genitourinary organs must be treated as a generalized disease. It must be assumed that an active primary focus also exists.

Triple drug therapy is indicated for a minimum of 2 years: (1) Streptomycin Sulfate, U.S.P., 1 Gm. I.M. twice weekly; (2) Amino-salicylic Acid, U.S.P. (PAS), 8-12 Gm. orally/day in divided doses; and (3) Isoniazid, U.S.P. (INH), 3-5 mg./Kg./day in divided doses.

If no gross pyelographic change is present, nephrectomy is not indicated. Bilateral infection can only be treated by medical measures. However, if only 1 kidney is involved and shows definite ulceration of 1 or more calyces, nephrectomy should be done after 2-4 months of medical treatment. The urine must be examined carefully by stain of sediment, culture, and guinea pig inoculation for Mycobacterium tuberculosis for years, since relapse is not uncommon.

Vesical infection usually responds well to antimicrobial therapy. If severe vesical contracture occurs, diversion of the urine (e.g., ureterosigmoidostomy) or ileocystostomy (to increase bladder capacity) may be necessary.

Prognosis.
If the disease is limited to 1 kidney and if the bladder and seminal tract are not involved, the outlook is excellent.

CHEMOTHERAPEUTIC AND ANTIBIOTIC TREATMENT OF UROLOGIC INFECTIONS

Choice and Dosage of Drugs. (See p. 614.)
In order to cure infections of the urinary tract, particularly those in the chronic stage, standard therapeutic dosages of anti-infective drugs should be administered for at least 2 weeks and preferably longer. In order to suppress an incurable infection, one-half to one-fourth of this dose should be administered for many months or years.

The most useful drugs in the treatment of nonspecific infections are the sulfonamides. Most acute infections respond to them; if they do not, a chronic infection or urinary stasis may be present and further examination is required.

The tetracyclines and chloramphenicol are the most useful antibiotics and should be used after urine has been obtained for culture and sensitivity tests in the very ill patient, in the patient who has not responded to a sulfonamide, or a patient suffering from a refractory chronic infection.

Despite prolonged use of the most appropriate antibiotic, long-standing chronic infection is usually refractory. Suppressive therapy for months or years should then be instituted. Small doses of a sulfonamide or one of the urinary antiseptics (e. g., nitrofurantoin, methenamine mandelate) are effective. Since measurable blood or tissue levels cannot be achieved with urinary antiseptics, their initial use in the treatment of acute or chronic infections is not recommended.

URINARY STONES

Urinary lithiasis may occur in association with metabolic diseases (e. g., cystinuria, gout, hyperparathyroidism) or can be secondary to infection. Many cases are idiopathic. Calculi are most common in men.

RENAL AND URETERAL CALCULI

Clinical Findings.
A. Symptoms and Signs: Nonobstructive stones usually cause no symptoms. A small stone in the ureter or kidney is apt to cause agonizing renal pain and colic radiating along the course of the ureter. Gross hematuria is not uncommon.

Tenderness over the kidney may be noted. Marked abdominal distention due to paralytic ileus is a common secondary finding in patients with ureteral stone.
B. Laboratory Findings:
1. Urinalysis - Red cells are commonly found, white cells and bacteria may be present. Oxalate, phosphate, uric acid, or cystine crystals may be seen.

A Sulkowitch test (see p. 373) may reveal hypercalciuria, which suggests hyperparathyroidism. If the stone is only mildly radiopaque, if there have been many recurrences, or if there is a family history of lithiasis, a qualitative test for cystine in the urine should be done.

On a low-calcium diet (no dairy products), the total urinary calcium should not exceed 175 mg./24 hours. Higher values suggest hyperparathyroidism or idiopathic hypercalciuria.
2. Renal function tests - The PSP will be normal unless there is bilateral obstruction or infection.
3. Special blood chemistry studies - Fasting serum calcium, phosphorus, and proteins should be determined, particularly if the Sulkowitch test is strongly positive. Determination of the tubular reabsorption of phosphate may be indicated; it is low (40-80%) in hyperparathyroidism.

Serum uric acid tests may reveal evidence of gout and suggest uric acid stone.
4. Stone analysis - Stones passed previously should be analyzed. This is important when outlining prophylactic treatment.

C. X-ray Findings: A KUB will afford presumptive evidence of stone since 90% are radiopaque. Excretory urograms accurately localize the radiopaque body in the urinary tract and depict the degree of obstruction or renal damage caused by the stone.

D. Instrumental Examination: Cystoscopy for diagnostic purposes is seldom necessary if excretory urograms are satisfactory.

Differential Diagnosis.

Urography differentiates lithiasis from acute pyelonephritis and renal or ureteral tumor. Bacteriologic examination differentiates from renal tuberculosis.

Complications.

Infection often develops secondary to a renal or ureteral stone. Obstruction at the ureteropelvic junction or in the ureter, caused by a stone, leads to progressive hydronephrosis.

Treatment.

A. Conservative Treatment:
 1. Renal stone - No treatment is indicated for pelvic or calyceal stones, which cause few or no symptoms and produce no obstruction. Most staghorn calculi are best left alone since they are usually nonobstructive and seldom cause pain.
 2. Ureteral stone - The majority of stones that reach the ureter will pass by themselves.

B. Cystoscopic or Surgical Measures:
 1. Renal stone - If a renal stone is obstructive or is associated with disabling pain or recurrent infection, it should be removed surgically. Nephrectomy may be necessary if obstruction and infection have markedly impaired renal function.
 2. Ureteral stone - Cystoscopic manipulation or ureterolithotomy is necessary if progressive hydronephrosis develops, if persistent infection supervenes, or if the stone is judged to be too large to pass spontaneously.

Prophylaxis.

A. Specific Measures:
 1. Calcium stones - Primary hyperparathyroidism requires parathyroidectomy. Eliminate milk and cheese from the diet, since these comprise the major source of calcium. Should the Sulkowitch test remain strongly positive, give sodium phytate to absorb the calcium in the intestine.
 2. Phosphate stones - Acidification of the urine markedly increases the solubility of calcium or magnesium ammonium phosphate. The amount of phosphate excreted in the urine may be decreased by the administration of aluminum gels, which absorb phosphate in the intestinal tract.
 3. Oxalate stones - A low-oxalate diet may decrease the amount of oxalate in the urine.
 4. Metabolic stones - These include cystine and uric acid stones, both of which are insoluble in acid urine; the urinary pH should be raised to 7.5 or higher in order to increase their solubility. In addition to an alkaline-ash diet, alkaline drugs (e.g., sodium and potassium citrate) are

usually necessary. It is advantageous to limit the purines
in the diet of uric acid stone formers. Severe cystinurics
will require a low-methionine diet (restriction of sulfur-
containing amino acids), since methionine is the precursor
of cystine.

B. General Measures: A large fluid intake will keep the solutes
dilute. Infection should be combatted; obstruction and stasis
should be corrected. Avoid prolonged recumbency.

Prognosis.

Renal stone recurs in a significant number of cases, and the
prognosis is therefore guarded. The patient must be followed
carefully for years. The real danger from stone is renal damage
caused by obstruction and infection.

VESICAL CALCULI

Vesical calculi almost always occur as a complication of other
urologic disease. Ninety-five percent occur in men. The most
common cause is vesical residual urine which is infected by urea-
splitting organisms.

Clinical Findings.
A. Symptoms and Signs: The patient may complain of sudden
interruption of the stream of urine and pain radiating down the
urethra as the stone rolls onto the bladder neck. Hematuria is
not uncommon.

B. Laboratory Findings: The urine is almost always infected.
Red cells are often noted. Excretion of PSP may be depressed
because of residual urine (see p. 374).

C. X-ray Findings: Vesical stones are usually visible on a plain
film.

D. Instrumental Examination: A catheter may be arrested by a
urethral stricture. If it passes to the bladder, it will usually
recover infected residual urine. A urethral sound passed to
the bladder may cause a "click" when it hits a stone. Cys-
toscopy will clearly visualize the calculi and help in the
diagnosis of the primary cause.

Differential Diagnosis.

Cystoscopy differentiates vesical stone from vesical tumor
and extravesical calcifications seen on x-ray.

Complications.

A small stone may become lodged in the urethra.

Treatment.
A. Small stones can be removed by cystoscopic means. Larger
ones can be crushed with the lithotrite transurethrally. If the
stone is very large, cystotomy may be necessary.

B. Treatment of Complications: Infection cannot be eradicated
until the stone and its cause are removed.

Prophylaxis.

Stasis and infection, whether primary or secondary, must be

eradicated. Early mobilization of injured patients will do much to prevent vesical calculi.

Prognosis.
    The rate of recurrence of vesical stone is low if the primary cause is successfully treated.

INJURIES TO THE
GENITOURINARY TRACT

INJURIES TO THE KIDNEY

Renal injuries are not common but are potentially serious and may be complicated by trauma to other organs or structures. The renal injury may range from a mild ecchymosis, bruise, or contusion to laceration or rupture.

Clinical Findings.
  A. Symptoms and Signs: Pain in the renal area may be obscured by the severity of other injuries. Hematuria is usually present. Shock or signs of hemorrhage may be present. Ecchymoses and tenderness over the renal area may be noted. A mass felt or percussed in the flank may represent hematoma, urinary extravasation, or both.
  B. Laboratory Findings: The Hct., when followed serially, is of the greatest importance; progressive anemia means hemorrhage. Red cells are usually found on urinalysis.
  C. X-ray Findings: A KUB may show grayness of the renal area and loss of the renal and psoas shadows if perirenal fluid (urine, blood) is present.
        As soon as shock has been treated, excretory urograms should be taken. If the urogram shows evidence of injury to 1 kidney (e.g., no visualization, extravasation), it is important to note that the contralateral kidney is normal since emergency nephrectomy might have to be considered.
  D. Instrumental Examination: Cystoscopy and ureteral catheterization are seldom necessary.

Differential Diagnosis.
    Trauma to the lumbar area and fractures of the ribs, spine, or transverse processes may cause local symptoms suggesting renal injury. In these injuries, hematuria is absent and urograms normal.

Complications.
    Excretory urograms should be obtained 3-6 months after any renal injury for the purpose of diagnosing ureteral stenosis with secondary hydronephrosis, or atrophy of the kidney due to injury or thrombosis of the renal artery. The latter complication may cause hypertension.

Treatment.
  A. Emergency Measures: Treat shock and hemorrhage.

B. Surgical Treatment: 10-20% of injured kidneys may require surgical intervention because of persistent bleeding. The laceration may be sutured, but nephrectomy may be necessary if injury is severe and the other kidney is normal.

C. Treatment of Complications: Perinephric infection requires surgical drainage. Late complications may indicate the need for nephrectomy or repair of ureteral stenosis.

Prognosis.

In contusions, the prognosis is excellent. When rupture occurs, serious complications may supervene.

INJURIES TO THE URETER

Ureteral injuries most often occur accidentally during difficult and extensive pelvic surgery.

Clinical Findings.

A. Symptoms and Signs: Persistence of abdominal distention and lack of peristalsis past the second or third postoperative day suggest the possibility of ureteral injury. Signs of peritonitis may be elicited. Urine may be seen draining from the wound or the vagina.

B. Laboratory Findings: Blood counts and urinalysis are of little value. The PSP may be depressed if 1 ureter is occluded.

C. X-ray Findings: A plain film of the abdomen may reveal grayness in the pelvis if extravasation has occurred. Excretory urograms may reveal extravasation. If the ureter has been completely occluded, the renal pelvis and calyces may receive no radiopaque fluid but progressive increase in density of the renal parenchyma may be noted. Retrograde ureterograms will reveal the site of obstruction.

D. Instrumental Examination: Ureteral catheterization will demonstrate patency or obstruction.

Differential Diagnosis.

Urinary specific gravity, PSP test, cystoscopy, and urograms differentiate bilateral ureteral injuries from acute renal failure.

Complications.

These include urinary fistula, ureteral stenosis with hydronephrosis, renal infection, and peritonitis.

Treatment.

Reanastomosis over a splinting catheter or "T" tube is highly successful. Reimplantation into the bladder – or a tube formed of adjacent bladder wall – usually affords a good result.

If the injury is discovered late and hydronephrosis is advanced, nephrectomy may be the operation of choice.

Prophylaxis.

The surgeon operating in the pelvis must identify the ureters in order to prevent ureteral injury. When difficult or extensive abdominal procedures are anticipated, indwelling ureteral catheters should be placed just prior to the operation for ease of identification.

Prognosis.
Prognosis is best when the ureter is repaired at the time of injury. Delayed repair is less successful because of the fibrous tissue reaction and renal damage from obstruction.

INJURIES TO THE BLADDER

The bladder may be injured by external forces or during surgery.

Clinical Findings.
A. Symptoms and Signs: Gross hematuria and suprapubic pain are
   to be expected. Shock and evidence of local trauma may be
   noted. Suprapubic and rebound tenderness are usually found.
   A large suprapubic mass may be felt. Rectal examination may
   reveal a large boggy mass above the prostate.
B. Laboratory Findings: Urinalysis reveals blood.
C. X-ray Findings: A KUB may reveal pelvic fracture. Increased
   grayness of the vesical area suggests extravasation. Excre-
   tory urograms will survey the kidneys for injury.
       A cystogram (see p. 374) is the most dependable test for
   vesical injury.

Differential Diagnosis.
Injury to other urologic organs is differentiated from vesical injury by appropriate x-ray technics.

Complications.
Rarely, the degree of hemorrhage may endanger life. Evidence of injury to other organs must be sought.

Treatment.
A. Emergency Measures: Treat shock and hemorrhage.
B. Specific Measures:
    1. Extraperitoneal rupture - Drain the site of injury surgically.
       Open the peritoneum and explore the intraperitoneal organs
       for injury. An indwelling urethral catheter should be placed.
    2. Intraperitoneal rupture - Close the tear transperitoneally
       if possible. Both an indwelling urethral catheter and a
       cystostomy tube should be utilized.

Prognosis.
If the diagnosis is made and proper treatment instituted within 6-12 hours, morbidity and mortality will be minimal. Delay may allow the development of severe cellulitis or peritonitis.

INJURIES TO THE MEMBRANOUS URETHRA

This portion of the urethra is enclosed in the ligamentous urogenital diaphragm attached to the pubic bone. Fracture of this bone often results in tears of this portion of the urethra.

Clinical Findings.
A. Symptoms and Signs: Urethral bleeding is common. The

patient may be unable to void. Pain may be present in the lower abdomen or perineum. A tender suprapubic mass (blood, urine, or both) may be felt. Rectal examination may reveal a large boggy mass; the prostate may be avulsed and dislocated upward.

B. Laboratory Findings: The urine is usually grossly bloody.

C. X-ray Findings: A plain x-ray may show fracture of the pelvic bones. A urethrogram (see p. 374) may reveal the site of injury. If a catheter can be passed to the bladder, a cystogram should be done to rule out vesical damage.

D. Instrumental Examination: A catheter may be arrested at the site of injury.

Differential Diagnosis.

Other urologic injuries are differentiated by appropriate x-ray technics.

Complications.

Hemorrhage and urinary extravasation are the serious early complications. The common late complication is urethral stricture. Impotence is not uncommon.

Treatment.

A. Emergency Measures: Treat shock and hemorrhage.

B. Specific Measures: If a catheter can be passed to the bladder it should be left in place for 14-21 days. If a catheter is arrested at the site of injury, suprapubic repair over a Foley catheter should be done. One lb. of traction should be placed on the end of the catheter to approximate the ends of the urethra. The area should be drained.

C. Treatment of Complications: Periodic urethral sounds (to 24 F.) are indicated for at least 1 year to prevent stricture formation (see p. 402).

Prognosis.

The immediate mortality is low. Stricture may develop.

INJURIES TO THE BULBOUS URETHRA

Straddle injuries to the bulbous urethra cause bleeding and urinary extravasation which may spread to the scrotum and penis. There is local pain. Urination may cause extravasation. Urethral bleeding is common. A mass is present in the perineum. Ecchymosis of the penile and scrotal skin appear later.

Hematuria is present. The WBC may be elevated if infection ensues. A urethrogram (see p. 374) will demonstrate the site and degree of injury. With minor injuries, a catheter may pass to the bladder. Arrest of the catheter suggests the presence of more serious damage.

The early complications are perineal hematoma and urinary extravasation. The late complication is urethral stricture.

If the patient can void and there is no urinary extravasation, no treatment is necessary. In more severe injuries, the passage of a catheter should be attempted. If this is successful, the catheter

should be left indwelling for 3 weeks. If not successful, surgical repair over a splinting catheter is necessary.

If perineal urinary extravasation and hematoma formation are extensive, drainage may be necessary.

All patients suffering from urethral injury require periodic sounding to prevent stricture formation.

INJURIES TO THE PENDULOUS URETHRA

The pendulous urethra is most commonly injured by instruments used for the treatment of severe urethral stricture. Some urethral bleeding is to be expected. If the injury has been severe, the catheter may be arrested.

If urination is unaffected, no treatment is necessary. Severe bleeding may require the placement of a large catheter. In more severe injuries, an indwelling catheter should be left in place for 14 days. Surgical repair (over a catheter) may rarely be necessary.

Postoperatively, sounds should be passed periodically. The only sequel is persistent urethral stricture.

TUMORS OF THE
GENITOURINARY TRACT

Neoplasms of the prostate gland, bladder, and kidneys are among the most common abnormal growths. Tumors of the testis are highly malignant and afflict young men. Neoplasms of other genitourinary organs are rare.

ADENOCARCINOMA OF THE KIDNEY
(Grawitz's Tumor; Hypernephroma)

About 80% of renal neoplasms are adenocarcinomas, and two-thirds of these occur in men. They apparently arise from adenomas or renal tubule cells.

Most metastases occur by way of the blood stream to the liver, lungs, and the long bones. Regional lymph nodes and at times the supraclavicular nodes may also be involved.

Clinical Findings.
  A. Symptoms and Signs: Painless total gross hematuria is the most common symptom. An abdominal mass noted by the patient may be the presenting complaint.

   A mass in the flank may be found. It is usually firm and is often nodular.
  B. Laboratory Findings: The cardinal sign is microscopic or gross hematuria. Erythrocytosis has also been reported.
  C. X-ray Findings: A KUB may show an enlarged kidney. Excretory urograms show calyceal distortion from a space-occupying mass. If these films are not diagnostic, retrograde urograms may be necessary.
  D. Instrumental Examination: If the patient has gross hematuria

when seen, immediate cystoscopy is mandatory. Bloody efflux from 1 ureteral orifice is an invaluable sign.

Differential Diagnosis.
Urography differentiates this tumor from hydronephrosis, polycystic disease, and extrarenal tumors.

Complications.
These include metastases and hydronephrosis from ureteral compression.

Treatment.
Nephrectomy is indicated in the absence of metastases. Although these tumors are radioresistant, irradiation may be utilized for local recurrence or osseous metastases.
About 25% of patients are alive 5 years after nephrectomy.

EMBRYOMA OF THE KIDNEY
(Wilms's Tumor; Adenomyosarcoma)

Embryoma is a highly malignant mixed tumor seen almost exclusively before the age of 6 years. Five per cent are bilateral. The sites of metastases are the lungs, liver, and brain. The common symptom is the discovery of a mass in the flank. Late symptoms include anorexia and loss of weight. Hypertension is present in 50% of cases. Hematuria is rare. Total renal function is usually normal. Urograms will show an enlarged kidney with calyceal distortion. Cystoscopy and ureteral catheters will afford retrograde urograms where the excretory urograms are equivocal.
Urography differentiates this tumor from neuroblastoma of the adrenal, hydronephrosis, and polycystic kidneys.
Immediate nephrectomy is essential unless metastases are found. Postoperative irradiation should be given. Radiation therapy should be administered, even if metastases have occurred, since these tumors are exceedingly radiosensitive.
About 30% of children treated before metastases have occurred can be cured. The younger the child, the better the prognosis.

TUMORS OF THE RENAL PELVIS AND URETER

Histologically, tumors of the renal pelvis and ureter resemble tumors of the bladder (see p. 393). They may be benign or malignant. The pelvic tumors comprise 10% of renal neoplasms. Ureteral tumors are relatively rare. Secondary similar tumors are apt to be found distal to the primary growth (including the bladder).
Metastases involve principally the regional lymph nodes.
Hematuria is the commonest complaint. There may be pain if the tumor is obstructive or if clots pass down the ureter. Red cells are commonly found in the urine. An obstructive tumor may lead to infection. Excretory urograms will disclose a space-occupying lesion in the pelvis or ureter. Retrograde urograms may be indicated if excretory urograms are equivocal. Cystoscopy may reveal blood emanating from a ureteral orifice. Search should be made

for "satellite" vesical tumors. A Papanicolaou smear may reveal tumor cells.

Adenocarcinoma of the kidney (see p. 391) may also cause hematuria. Urograms should establish the diagnosis.

Nonopaque stone (see p. 384) can mimic pelvic or ureteral tumor on urograms. The differentiation may only be made at surgery.

Unless metastases are present, complete ureteronephrectomy with removal of a cuff of periureteral bladder wall is indicated.

With benign tumors the prognosis is excellent. The outlook for patients with well-differentiated tumors is fair, those with anaplastic neoplasms usually die of metastases within 2 years.

TUMORS OF THE BLADDER

Vesical tumors are relatively common. At least 75% occur in men after the age of 50. The cause is not known, but there is increasing evidence that carcinogens in the urine are etiologic factors.

Clinical Findings.
  A. Symptoms and Signs: Hematuria is the most common symptom. Secondary infection (see p. 379) may develop. Perivesical spread causes constant suprapubic pain.

  There is usually little to be found on abdominal examination unless a large tumor is present. On rectal or vaginal examination, fixation and induration may be noted at the base of the bladder.
  B. Laboratory Findings: Secondary infection of the urine is common. Bleeding is apt to be intermittent, and for this reason red cells may not always be found on urinalysis. Renal function tests are normal unless bilateral ureteral obstruction has developed.
  C. X-ray Findings: Excretory urograms may show ureteral obstruction. On the cystogram, a space-occupying lesion may be noted. Careful search for a primary renal pelvic or ureteral tumor should be made.
  D. Instrumental Examination: Cystoscopy makes the diagnosis.
  E. Cytology: A Papanicolaou smear may reveal malignant cells.

Differential Diagnosis.
  Hematuria due to other causes is differentiated by appropriate diagnostic technics (e.g., cystoscopy, urography).

Complications.
  Secondary infection of the bladder is common. Hydronephrosis may occur as a result of ureteral occlusion. Urinary retention may supervene if the tumor occludes the bladder neck.

Treatment.
  A. Specific Measures: Transurethral resection will cure the less invasive lesions and control some of the less well-differentiated growths. Cystectomy is indicated for the more invasive tumors without demonstrable metastases. This requires urinary diversion (e.g., ureterosigmoidostomy).
  B. Palliative Procedures: Radiation therapy may control the more

anaplastic tumors and prevent the development of new papillomatous lesions.

C. Treatment of Complications: Control of secondary infection should be attempted (see p. 379). Nephrectomy may be indicated for an infected hydronephrotic kidney.

Prognosis.

The well-differentiated papilloma may recur or new tumors may form; periodic cystoscopy is therefore imperative. Later recurrences may be less well-differentiated and more malignant.

The prognosis for sessile infiltrating carcinomas is poor even though radical treatment is employed.

BENIGN PROSTATIC HYPERPLASIA

The term "benign prostatic hyperplasia or hypertrophy" is a misnomer since it is not the prostate itself but the prostatic periurethral glands which undergo hyperplasia and in so doing compress the true prostate laterally to form the "surgical" capsule.

Clinical Findings.
A. Symptoms and Signs: The syndrome of "prostatism" includes hesitancy and straining in initiating urination, loss of force and caliber of the stream, terminal dribbling, and (usually) frequency and nocturia. The degree of the latter 2 symptoms depends upon the volume of residual urine and the presence or absence of complicating infection. Rupture of dilated veins at the bladder neck may cause hematuria. Acute urinary retention may develop.

A suprapubic mass may be found if a great deal of residual urine is present. The prostate may or may not be enlarged and may be of any consistency from soft to firm. (Stony hardness means cancer until proved otherwise.)

B. Laboratory Findings: Urinalysis may reveal infection. If the first half-hour PSP specimen contains 50-60% of the dye, there is no residual urine. If less than this is excreted, there is either residual urine or impaired renal function.

C. X-ray Findings: Excretory urograms may reveal complicating hydronephrosis or vesical calculi.

D. Instrumental Examination: Catheterization immediately after voiding will measure residual urine. Cystoscopy will reveal the degree of prostatic enlargement and the secondary changes in the bladder wall (e.g., trabeculation).

Differential Diagnosis.
Benign prostatic hypertrophy must be differentiated from neurogenic bladder and cancer of the prostate.

Complications.
Obstruction leads to infection of the bladder, kidneys, or prostate. Vesical calculi (see p. 386) may form.

The obstruction may cause vesical diverticula. Hydronephrosis may occur when a ureterovesical valve becomes incompetent.

Treatment.
  A. Conservative Treatment: Mild to moderate prostatism may be
     considerably relieved by 3-4 prostatic massages given every 2
     weeks. Massages are of value also in reducing edema due to
     prostatitis, if present. Secondary infection (see Cystitis, p.
     379) should be treated with antimicrobial drugs.
         The patient should be cautioned to void as soon as the desire
     is noted and not to drink a large volume of fluid in a short time.
         Catheterization is mandatory for acute urinary retention.
     If spontaneous voiding does not then occur, a catheter may be
     left indwelling for 3 days. The indwelling catheter may be the
     treatment of choice for the poor-risk patient.
  B. Surgery: Four procedures are available: (1) transurethral
     prostatectomy for the smaller glands; (2) suprapubic; (3) retro-
     pubic; and (4) perineal prostatectomy, for the larger glands.

Prognosis.
    Surgical removal of the obstructing tissue has a low mortality
and should relieve most symptoms.

CARCINOMA OF THE PROSTATE

    Cancer of the prostate is rare before the age of 60. The cause
is not known, but growth of the neoplasm is influenced by sex hor-
mones.
    Most cancers arise in the "surgical" capsule (the true pros-
tatic tissue). The tumor finally grows into the seminal vesicles
and surrounding tissues.
    These malignancies spread via the perineural lymphatics to the
regional lymph nodes. Metastases also occur through the veins,
particularly the vertebrals, and most commonly involve the bones
of the pelvis. Visceral spread is also seen.

Clinical Findings.
  A. Symptoms and Signs:
     1. Early - The presenting symptoms are usually from obstruc-
        tion, and are similar to those described in the discussion of
        benign enlargement (see p. 394). Rectal examination dis-
        closes a firm, usually flat plaque in the prostatic capsule.
        It is usually not raised above the surface of the gland.
     2. Late - There may be complaints referable to local spread or
        metastases: pain in the low back radiating down 1 or both
        legs, edema of 1 or both legs, enlarged supraclavicular
        nodes, and loss of weight. The entire gland is usually stony
        hard and often nodular and fixed. The seminal vesicles may
        be involved.
  B. Laboratory Findings: Anemia may be extreme if bone marrow
     is replaced by tumor. Urinalysis may show infection. In the
     early stages of obstruction, renal function is unimpaired. With
     bilateral ureteral obstruction, renal function will be depressed.
         Serum acid phosphatase is often increased when local ex-
     tension or metastases have occurred. Serum alkaline phos-
     phatase may also be elevated when bony metastases develop.
  C. X-ray Findings: X-rays of the pelvic bones may reveal typical
     osteoblastic metastases.

D. **Instrumental Examination:** Catheterization will measure the residual urine. Cystoscopy will reveal the degree of obstruction and the secondary changes in the bladder wall.

E. **Biopsy:** If the lesion is extensive and incurable, perineal needle biopsy is indicated. The tissue removed on transurethral resection also affords tissue for pathologic diagnosis.

Differential Diagnosis.

Benign prostatic hyperplasia can usually be differentiated by palpation of the prostate. Osteoblastic metastases or elevation of serum acid phosphatase establishes the diagnosis of cancer.

Benign prostatic nodules may be caused by tuberculosis, chronic prostatitis, or prostatic calculi. Biopsy may be necessary to differentiate these lesions from cancer.

Complications.

In addition to the changes secondary to the obstruction (see p. 375), spontaneous fractures may occur at the sites of metastases.

Treatment.

A. **Specific Measures:** Any prostatic nodule which may be carcinoma should be biopsied; if pathologic examination is positive, radical prostatectomy should be done.

B. **Palliative Measures:**

1. Antiandrogen therapy (for men whose tumors are too advanced for radical surgery) - One of the following is indicated:
   a. Estrogen medication - Diethylstilbestrol, 5 mg. daily.
   b. Orchiectomy.
   c. Medical adrenalectomy by administration of corticosteroids may afford some comfort for a few months when antiandrogen therapy fails.

2. Transurethral resection may be necessary if vesical neck obstruction is severe.

Prognosis.

Radical prostatectomy will cure half of cases when the lesion is localized. Palliative treatment affords considerable temporary relief for most patients. The majority die within 3 years; a few may be controlled for 10 years or more.

TUMORS OF THE TESTIS

With rare exceptions, all testicular tumors are malignant. Most occur in men between the ages of 20 and 35 years. The most common type is the seminoma. Second in incidence is carcinoma. Many of these tumors elaborate chorionic gonadotropins; this finding is usually associated with hyperplasia of the Leydig cells. The true teratoma is composed of both epithelial and mesenchymal components.

Interstitial cell tumors are rare and benign. They secrete both androgen and estrogen, which causes precocious sexual maturation in boys and gynecomastia in men. The rare Sertoli cell tumor is benign and feminizing; gynecomastia is the rule.

The common sites of metastases are the pelvic and preaortic lymph nodes, lungs, and liver.

Clinical Findings.

A. Symptoms and Signs: The most common symptom is painless enlargement of a testicle. Gynecomastia may be noted with those tumors which elaborate gonadotropins. Symptoms due to metastases include an abdominal mass, pain from bowel or ureteral obstruction, and loss of weight.

The testis is usually enlarged, firm, smooth, and heavy. Squeezing such a lesion fails to elicit typical testicular pain. Ten per cent are associated with secondary hydrocele.

B. Laboratory Findings: The presence of urinary chorionic gonadotropins means that a carcinoma or chorio-epithelioma is present. Urinary 17-ketosteroids are elevated with Leydig cell tumor. Urinary estrogens may be increased with both Leydig and Sertoli cell tumors.

C. X-ray Findings: Excretory urograms may show displacement or obstruction of the ureter caused by involved lymph nodes.

Differential Diagnosis.

Hydrocele (see p. 404) is cystic and transilluminates. Spermatocele (see p. 404) is a cyst which lies free above the testis. It contains sperm. Gumma of the testis is rare. A positive serologic test for syphilis should suggest the diagnosis.

Complications.

The complications arise from metastases. A ureter may become occluded by pressure from involved lymph nodes.

Treatment.

If testicular tumor cannot be ruled out, orchiectomy should be done through an inguinal incision.

Radical retroperitoneal lymph node resection is indicated for all but seminoma and chorio-epithelioma. It is contraindicated when gross metastases are demonstrated.

X-ray therapy to the regional lymph nodes (iliac and preaortic) is indicated in all cases, seminomas are quite radiosensitive.

Prognosis.

The prognosis is poor if there are obvious metastases, chorionic gonadotropins in the urine (choriocarcinoma), or hyperplasia of the interstitial cells. Seminoma offers the best prognosis; chorio-epithelioma the poorest.

TUMORS OF THE PENIS

Tumors of the penis are epidermoid in type. The majority arise on the glans under a redundant foreskin. Metastases involve the subinguinal, inguinal, and later the iliac nodes.

Biopsy is mandatory for all suspicious penile lesions. Dark-field examination will differentiate tumor from chancre.

Tumors of the penis require amputation at least 1.5 cm. proximal to the growth. In the absence of lymphadenopathy, lymph node dissection is not necessary.

The prognosis is good in small epitheliomas. It is poor when lymph nodes are extensively involved.

DISORDERS OF THE PERIRENAL AREA

Disorders of the perirenal area include enlargements and tumors of the adrenal glands and connective tissue neoplasms. The majority involve the adrenal glands (e. g., tumors and hyperplasia), and most of these cause endocrinologic symptoms and signs (e. g., paroxysmal hypertension, virilism).

Diagnosis is corroborated and the tumor localized by the usual technics of urologic diagnosis. A KUB may reveal the mass. Urograms may more clearly delineate displacement of the kidney.

Retroperitoneal gas insufflation usually outlines the extrarenal mass. Gastrointestinal series and barium enema may demonstrate displacement of a portion of the enteric tract.

DISORDERS OF THE KIDNEYS*

POLYCYSTIC KIDNEY DISEASE

Polycystic kidneys are larger than normal. Cysts of various sizes are scattered throughout and cause progressive impairment of function. In severe cases, death occurs during infancy. The remainder present themselves for diagnosis and treatment after the age of 40 years.

Clinical Findings.
  A. Symptoms and Signs: The weight of the kidneys may cause pain. Hematuria is common. In the stage of renal insufficiency, complaints may include headache, nausea and vomiting, weakness, and loss of weight.

One or both kidneys may be palpable. Hypertension is often found. Fever may accompany pyelonephritis or infection of a cyst.
  B. Laboratory Findings: Anemia secondary to uremia may be found. Proteinuria and hematuria are the rule. Pus cells and bacteria are commonly present. The PSP will reveal some functional impairment.
  C. X-ray Findings: Both kidneys are usually significantly enlarged on the KUB. Urograms reveal marked bilateral calyceal distortion.
  D. Instrumental Examination: If renal function is poor, retrograde urograms may be required for diagnosis.

Differential Diagnosis.
Bilateral simple cysts may deform a few calyces, but calyceal distortion is more bizarre in polycystic disease.

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*See Index for Renal Infection, Tumors, etc.

Complications.
Pyelonephritis is a common complication.

Treatment.
A. General Measures: Give a low-protein diet, and force fluids so that urinary output is 1500 ml./day if there is impairment of renal function.
B. Treatment of Complications: Pyelonephritis must be rigorously treated (see p. 377).

Prognosis.
If the disease presents in childhood, the prognosis is poor. Patients in middle age are apt to die of uremia within 5-10 years after the diagnosis is made.

SIMPLE (SOLITARY) CYST

Simple cysts are the most common space-occupying lesions of the kidneys. They are usually unilateral and single. As such a cyst grows, it may destroy renal parenchyma. Calcification of the cyst wall is occasionally seen. About 10% contain hemorrhagic fluid; half of these have papillary tumors on their walls.

Flank pain may be present. Bleeding into a cyst may cause sudden severe pain. The cyst may be palpable. Urinalysis and renal function are usually normal. Urograms will show distortion of the adjacent calyces.

Renal carcinoma is differentiated from cyst by urinalysis (hematuria) and, at times, aortography, polycystic disease is differentiated by widespread bilateral calyceal distortion and depressed renal function.

Spontaneous infection of a simple cyst is rare. Sudden hemorrhage into a cyst causes severe pain. The bleeding may come from a complicating tumor on the cyst wall.

Surgical exploration is indicated because the differentiation between cyst and cancer of the kidney is difficult and because 5% of cysts have tumors on their walls. Treatment consists of excision of the extrarenal portion of the cyst.

Without complications, the prognosis is excellent if the cyst is excised. In untreated cases progressive damage to the kidney or malignant degeneration may occur.

RENAL FUSION

About 1 out of 1000 people have some type of renal fusion, the most common being the horseshoe kidney. Less frequently, the total renal tissue develops as 1 mass ("cake kidney") situated in the midline or in 1 flank. In either case, 2 separate ureters drain the kidney.

Most of these patients have no symptoms. A few may have ureteral obstruction secondary to aberrant vessels, which are always present. Hydronephrosis and secondary infection then develop.

Urograms will usually be diagnostic. The pelves of a horseshoe

kidney are situated on the anterior surfaces of the renal masses. The lower calyces extend onto the psoas muscles (in the isthmus). Two pelvicalyceal systems may be noted in the "cake" kidney with both ureters entering the organ.

No treatment is necessary unless obstruction is present.

RENAL HYPERTENSION

Most cases of hypertension are of unknown etiology. Coarctation of the aorta, polycystic kidneys, glomerulonephritis, and periarteritis nodosa are often accompanied by high BP.

Renal ischemia may cause hypertension. The etiologic factors include chronic pyelonephritis and stenosis of the renal artery or its branches. The onset of malignant hypertension developing before the age of 30 or after 50 suggests the presence of renal ischemia.

Urinalysis may reveal infection. Total renal function is usually normal. Excretory urograms may show changes compatible with chronic pyelonephritis or some delay in excretion with poor concentration of the radiopaque material. A difference of as little as 1 cm. in the length of 1 kidney shadow on x-ray should suggest renal artery stenosis.

Ureteral catheterization permits evaluation of each kidney separately. This should be preceded by high salt intake. Useful tests include PSP, sodium or chloride concentration, and measurement of urine volume. A significantly ischemic kidney will excrete no more than half the volume of urine and at least 20% less sodium, chloride, or PSP than its mate.

Aortography is indicated if renal ischemia is suspected. It will reveal the caliber of the renal arteries.

Treatment consists of nephrectomy if the affected kidney is atrophic. Endarterectomy or another acceptable vascular surgical procedure is indicated if the involved kidney is near normal size and stenosis of the renal artery is demonstrated.

A significant number of hypertensive patients may be cured of hypertension when renal ischemia is corrected or the atrophic kidney removed.

DISORDERS OF THE BLADDER

EXSTROPHY OF THE BLADDER

Exstrophy of the bladder is a complete ventral defect of the urogenital sinus and the overlying skeletal system. The posterior bladder wall is continuous with the abdominal skin. The rami of the symphysis pubis are widely separated. Hydronephrosis with infection is common. Treatment usually consists of diversion of the urinary stream (e.g., ureterosigmoidostomy) and excision of the bladder. The associated epispadias (see p. 402) must also be repaired.

CONTRACTURE OF THE BLADDER NECK

Contracture of the bladder neck, usually of congenital origin, leads to back pressure changes in the bladder, ureters, and kidneys (see Obstruction and Stasis, p. 375). Infection is therefore a common complication. A distended bladder may be found. Residual urine is present, and renal function is often depressed. Excretory urograms usually reveal hydronephrosis, and cystograms often show ureteral reflux.

Cystoscopy will reveal hypertrophy of the bladder wall as well as the stenotic bladder neck. Relief can be obtained by a suprapubic plastic operation or by transurethral resection. If upper tract changes are marked, cystostomy or even nephrostomy may be indicated.

The prognosis depends upon the degree of renal damage. Many of these children die in uremia.

VESICAL FISTULAS

The bladder may communicate with the bowel or vagina. Vesicointestinal fistulas are secondary to intestinal malignancy, tuberculosis, or diverticulitis. Vesicovaginal fistula usually occurs following a gynecologic procedure (e.g., Wertheim operation) or in the advanced stage of cervical carcinoma.

Vesicointestinal fistulas are distinguished by the fact that the patient may pass gas and feces with the urine. In vesicovaginal fistulas there is constant drainage through the vagina.

Treatment of vesicointestinal fistula usually requires proximal colostomy followed months later by resection of the primary lesion and closure of the bladder. Repair of vesicovaginal fistulas may be performed either vaginally or suprapubically, depending upon the site of the opening.

DISORDERS OF THE PENIS AND MALE URETHRA

POSTERIOR OR PROSTATIC URETHRAL VALVES

Posterior or prostatic obstructing valves consist of folds of mucosa on the floor of the prostatic urethra.

Symptoms are usually those of sepsis. A distended bladder may be felt. Anemia from infection or uremia is common. The PSP excretion is low, and nitrogen retention in the blood is usually found.

Excretory urograms will reveal marked changes in the bladder, ureters, and kidneys if there is sufficient function to excrete the iodide. A cystogram often reveals bilateral ureteral reflux. A urethrogram may delineate the urethral valves.

Cystoscopy will show trabeculation of the bladder wall and diverticula. The valves may be visualized.

Treatment consists of destruction of the valves. Ureteral

reflux may require prolonged urethral or suprapubic catheter drainage. For advanced upper tract dilatation, nephrostomy drainage may be necessary.

The majority of these children die ultimately in uremia.

HYPOSPADIAS

The hypospadiac penis presents ventral curvature distal to the urethral meatus, which is proximal to its usual position. The meatus may open as far back as the perineum. In the latter instances, the scrotum is bifid, and there may be a cyst of the prostatic utricle. Cryptorchism is not uncommon.

No surgical treatment is necessary unless the degree of curvature with erection precludes sexual intercourse or if the orifice is so far back that semen cannot be deposited deep in the vagina.

EPISPADIAS

Epispadias is less common than hypospadias but is more disabling because urinary incontinence is usually present. The urethra opens proximal to the glans but on the dorsum of the penis, usually at the abdominopenile angle. Dorsal curvature is the rule. It should be noted that epispadias is a mild degree of exstrophy (see p. 400).

Treatment consists of correction of the urinary incontinence, straightening of the penis, and urethroplasty. If urinary continence cannot be gained, diversion of the urine may be necessary.

PHIMOSIS

Phimosis is the inability to retract the foreskin over the glans. Treatment consists of circumcision, but if acute inflammation is present preliminary incision in the dorsum of the foreskin may be necessary.

PARAPHIMOSIS

In paraphimosis the foreskin, once retracted behind the glans, is prevented from being drawn forward again by a tight preputial ring. This leads to compression of blood vessels and therefore edema of the glans. A dorsal slit in the constricting ring should be made. Circumcision should be done later.

URETHRAL STRICTURE

Acquired urethral stricture is usually secondary to a "straddle' injury (see p. 390) or a late complication of gonorrhea. A severe stricture causes the changes typical of obstruction (see p. 375).

Clinical Findings.

A. Symptoms and Signs: There is loss of force and caliber of the

stream. Urinary retention may occur. Cystitis or pyelo-
nephritis may be present. Periurethral induration may be felt
at the site of stricture. An abscess may be noted.

B. **Laboratory Findings:** Pus and bacteria are often found in the
urine. The PSP excretion may be diminished if renal damage
or residual urine is present.

C. **X-ray Findings:** A urethrogram (see p. 374) will reveal the
degree and site of the stricture. Fistulas may be demon-
strated. Urograms may reveal urinary calculi or chronic
pyelonephritis (see p. 378).

D. **Instrumental Examination:** A catheter or sound of average size
(22 F.) will be arrested by the stricture. Cystoscopy will show
hypertrophy of the vesical muscle and signs of infection.

Differential Diagnosis.

Prostatic obstruction (see p. 394) may cause similar symptoms,
but in this instance a catheter passes to the bladder with ease.

Carcinoma of the urethra will be diagnosed by biopsy.

Complications.

Prostatitis, cystitis, pyelonephritis, and urinary stones are
common complications. Periurethral abscess may develop at the
stricture site and may rupture through the skin, causing a fistula.

Treatment and Prognosis.

If a 20 F. sound is arrested, attempts to pass a filiform should
be made. If this is successful, moderate dilatation can be accom-
plished with followers of increasing sizes. Further dilatations with
larger filiforms and followers can then be done every 3-4 days. As
an alternative, an indwelling catheter can be passed to the bladder
after the initial dilatation and replaced every 2-3 days with a
catheter of the next largest size.

The stricture must be dilated every 3-6 months or gradual
contracture will occur. In a few cases the urethra will be too
fibrous to permit dilatation; in these instances surgical repair will
be necessary.

Most strictures can be kept open by periodic dilatations. The
prognosis depends upon the secondary effects upon the kidneys.

DISORDERS OF THE TESTES,
SCROTUM, AND SPERMATIC CORD

CRYPTORCHISM

Strictly speaking, the cryptorchid testis is one which has been
arrested along the path of normal descent (e.g., in the inguinal
canal or prepubic area), whereas an ectopic testis is one which has
wandered off the path (e.g., to perineum or the root of the penis).
Ten per cent of newborns have cryptorchism, but most descend
spontaneously during the next few weeks. Three per cent are
cryptorchid at puberty.

The spermatogenic (but not the Leydig) cells are quite sensitive

pheochromocytoma (e.g., in renal failure), but false-negative tests are rare.

b. **Histamine test** - (Must be performed in a hospital, as it may produce serious reactions. Use only in the normotensive patient.) Prepare the patient as for the phentolamine test. Inject 25-50 mcg. of histamine base I.V. and record the BP at one-minute intervals for 15 minutes. The test is positive if the BP rises within 3 minutes by over 50 mm. Hg systolic and 25 mm. Hg diastolic. False-positive and false-negative results may occur. Phentolamine should be on hand during this test so that the BP can be reduced if it becomes alarmingly high.

2. **X-ray examinations** - Plain films of the abdomen and chest, intravenous urography, and tomography of the adrenal region are usually adequate. Presacral oxygen injection may occasionally be of value. Aortography has been used but is dangerous.

Treatment.

Both adrenals should be explored as well as the sympathetic chain. Before and during the operation, hypertension is controlled by repeated injection of 5-75 mg. increments of phentolamine I.V. After removal of the tumor, profound hypotension may occur and can be counteracted by an intravenous infusion containing 8 mg. (or more if necessary) of norepinephrine in 1000 ml. of 5% dextrose in water. Norepinephrine drip may be required for 24-36 hours postoperatively to maintain the BP. If both adrenals have been partially or completely excised, adrenal cortical insufficiency should be combatted by administering hydrocortisone sodium succinate (Solu-Cortef®), 100 mg. I.V. every 4 hours for 3 doses, and then gradually reducing the dosage until it can be determined whether the patient has adrenal insufficiency.

In patients with nephrosclerosis the BP may not return to normal after removal of the tumor. The normotensive patient cannot be considered "cured" until a normal repeat histamine test or catecholamine determination has demonstrated that a second tumor is not present.

CUSHING'S SYNDROME
(Adrenocortical Hyperfunction)

The metabolic and clinical changes in Cushing's syndrome are caused by an excessive secretion of glucocorticoids, chiefly hydrocortisone. The hypersecretion may be due to tumor (benign or malignant), or, more commonly, to hyperplasia of the adrenal cortex. Cushing's syndrome is most common in women between 30 and 45 years of age.

Clinical Findings.
A. Symptoms and Signs: Weakness, menstrual irregularity or amenorrhea, moon facies, hirsutism, plethora, hypertension, truncal obesity, back pain, acne, psychiatric disturbances, easy bruisability, purple striae, increased insulin resistance, and impotence or loss of libido are the most characteristic

manifestations.

B. Laboratory Findings: Increased urinary levels of 17-hydroxy-corticoids are the most reliable indication of hypercorticism. In patients with normal levels, high levels may occur following an infusion of corticotropin. Urinary ketosteroids are variable: low levels suggest benign tumor; moderately elevated levels suggest hyperplasia; and very high levels of 17-ketosteroids (and 17-hydroxycorticoids) suggest cancer. Other laboratory findings which are frequently present are diabetic response to glucose tolerance tests, eosinopenia, leukocytosis, lympho-penia, alkalosis, and polycythemia.

C. X-ray Findings: The spine and long bones frequently show osteoporosis. Compression fractures of the spine, with de-mineralization, may occur. Rib fractures with callus forma-tion may be seen. The pituitary fossa is almost never enlarged preoperatively, but may be enlarged after surgery. The adrenal areas may be examined by oxygen insufflation (especially with tomograms) as for pheochromocytoma (see p. 404.1).

Treatment.

Tumors causing Cushing's syndrome are excised; hyperplasia requires total bilateral adrenalectomy. Pituitary irradiation (e.g., by implantation of radioactive isotope) is an alternate form of treatment in this latter group. Preoperative preparation (if renal function is adequate) consists of the administration of potassium chloride, 3-6 Gm. (45-90 gr.) daily orally for 1 week before sur-gery. A drip of 100 mg. of hydrocortisone sodium succinate (Solu-Cortef®), or its equivalent, in 500 ml. of isotonic saline solution (plus potassium) is begun at the start of the operation; 100 mg. by slow drip I.V. are given every 6 hours postoperatively for 24 hours; and the dosage is then gradually reduced. Unilateral tumors cause atrophy of the contralateral adrenal. Desoxycorti-costerone Acetate, U.S.P., 5 mg. I.M., is given on the first day and gradually reduced to 1-3 mg. daily as needed to overcome hypotension and hyponatremia. It is better to overtreat than under-treat with steroids during the immediate postoperative period. During the first 7-10 days after surgery, cortisone is decreased to a maintenance dose of 25-50 mg. orally daily. Totally adrenalec-tomized patients require permanent replacement therapy. After excision of a tumor, cortisone is gradually omitted. In the post-operative period, acute adrenocortical insufficiency may cause tachycardia, hypotension, fever, and often apprehension, disorien-tation, or coma. These findings are usually an indication for in-creasing the replacement therapy. Cortical insufficiency may • persist for months, and requires replacement therapy until the atrophic adrenal recovers.

The majority of functioning adrenocortical tumors are benign. Their prognosis is usually good. However, it is not always pos-sible to be certain that the lesion is benign, and subsequent follow-up is necessary. Since hypersecretion of steroids in the urine may be the earliest clue to metastases, urinary steroid measurements should be made every 6 months for several years after removal of a functioning tumor.

ADRENOGENITAL SYNDROME

The adrenogenital syndrome (adrenal virilism) is produced by adrenocortical hypersecretion of androgen. It may be secondary to cortical hyperplasia or benign or malignant tumor; or the adrenal glands may appear normal. Virilism may also be produced by adrenal rests in the ovaries, testicular tumors, and hypothalamic lesions.

In children, adrenal virilism produces precocious puberty. Axillary and pubic hair appear early. Height is increased at first, but these children will eventually be shorter than normal. In the female one finds pseudohermaphroditism; in the male, macrogenitosomia praecox. In the adult female there is enlargement of the clitoris, decreasing size of the breasts, male hair distribution and muscular development, acne, deepening of the voice, and amenorrhea.

The diagnosis is established by an increase in excretion of 17-ketosteroids in the urine. The region of the adrenals should be studied radiologically for tumor. If urinary 17-ketosteroids can be reduced to normal by prednisone, then the hypersecretion is due to hyperplasia and not to tumor. Hyperplasia should be treated by administration of prednisone or prednisolone, 5-25 mg. daily in divided doses. If tumor is demonstrated radiologically, or cannot be ruled out, exploration of the adrenals is indicated. Most virilizing adrenal tumors are malignant and are likely to have a poor prognosis.

It is important to make the diagnosis early. Pseudohermaphrodites should be raised as girls and treated with cortisone, clitoridectomy, and, later, plastic vaginal repair.

Feminizing tumors of the adrenal cortex are quite rare, are usually malignant, and are associated with increased amounts of estrogenic substances in the urine.

HYPERALDOSTERONISM

Excessive production of the mineral-regulating adrenal steroid, aldosterone, may be secondary to adrenal cortical hyperplasia or may be caused by tumor. This syndrome is characterized by muscular weakness, chronic hypertension, hypokalemia, hypernatremia, metabolic alkalosis, and associated carpal and pedal spasm and positive Trousseau and Chvostek signs. Polyuria, particularly nocturnal, and polydipsia are consistent findings. Carpal spasm may occur when the BP cuff is inflated and should immediately suggest this disorder. The urine is alkaline and of low specific gravity. The diagnosis is confirmed by finding an elevated urinary aldosterone excretion. Be certain the patient has not been on a salt-restricted diet and has not received chlorothiazide (Diuril®) for at least 2 weeks prior to the test.

Tumors are treated by excision, and hyperplasia by subtotal adrenalectomy. Give potassium preoperatively. Removal of most or all of the adrenal tissue calls for supportive therapy with corticosteroids.

Pyelonephritis is common in these patients. Hypertension may persist after surgery, and postoperatively these patients are very sensitive to mineral corticoids.

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Vascular Surgery

CONGENITAL VASCULAR DEFECTS

CONGENITAL ARTERIOVENOUS FISTULA

The multiple embryonic communications between the arterial and venous trunks occasionally persist as 1 or more direct or indirect connections between the artery and vein. These fistulas may not be clinically apparent for years.

The lower extremity is most frequently involved, but the vessels of the arm, neck, head, and brain are sometimes affected. Lesions in the extremities are most frequently around the joints. Multiple communicating channels, often involving long segments of vessels, make the condition difficult and often impossible to treat.

Lesions may exist as (1) lateral communications between the artery and vein, without altering the continuity of the main vessels; or (2) 1 or more arteries terminating in a plexus of veins. Capillary and cavernous hemangiomas are closely related disorders.

Clinical Findings.

Many of the local and systemic alterations noted in acquired arteriovenous fistulas are frequently present in the congenital form also (see Chapter 7, Pediatric Surgery).

A. Symptoms and Signs: The involved artery and vein become enlarged, tortuous, and thin-walled to a degree depending upon the number and size of the communications. The skin temperature is higher over the fistula. Very occasionally a thrill may be felt and a bruit heard. There may be increased hair and sweating in the region of the fistula, and the bones of the involved extremity may be abnormally long or eroded. Birth-marks in the area are common.

Chronic venous obstruction secondary to the fistula may manifest itself by varicose veins and stasis pigmentation, and distal ulceration may occur. Cardiac dilatation may eventually appear, although it is less frequent and pronounced than in the acquired form where the parasitic circulation through the fistula is usually larger.

B. Laboratory Findings: The oxygen saturation of the venous blood in the extremity with the fistula is higher than that of samples taken from the normal side.

C. X-ray Findings: Angiograms before or during surgery will sometimes give useful information, although the results are often confusing.

Treatment.

A. Conservative Treatment: When the communications are present in the lower extremities, pressure on the superficial veins by elastic bandages or stockings make it more difficult for the

blood to flow from the artery into the superficial veins. The
secondary varicose veins are at least partially controlled.
B. Surgical Treatment: Direct surgical correction is seldom pos-
sible, and even when attempted it may have to be done in stages.
Proximal ligation of the largest artery involved is not success-
ful; the communications themselves (they are usually multiple)
must be dissected out and ligated. In the rare case of single
fistula or where the abnormalities are confined to a limited
and readily accessible area, surgical correction by dividing the
communications is of value.

VASCULAR INJURIES

ACUTE ARTERIAL INJURIES

The diagnosis of vascular injury may be difficult, but an arter-
ial injury is usually present if the following findings are present:
The injured extremity is pale, waxy, mottled, cold, or cyanotic.
No pulsations can be felt distal to the injured area. (Distal pulsa-
tions do not rule out a vascular injury, however.) Varying degrees
of sensory loss may be present, from hypesthesia to anesthesia.
External hemorrhage may or may not be present; if it is not, the in-
jured limb is often much larger than the uninjured extremity. Is-
chemia of the muscles distal to the injury will usually cause severe
pain and partial or complete loss of muscle function.

A cold, pulseless limb may also be the result of shock, crush-
ing or contusing blows, or exposure. Accurate diagnosis may not
be possible until debridement is undertaken, although a wound in the
region of a large vessel should always arouse suspicion of an arter-
ial injury.

Treatment.
Regardless of the type of injury, the control of hemorrhage,
adequate blood replacement, and restoration of arterial blood flow
as soon as possible are the major objectives in management.

The best results are obtained when blood flow is reestablished
by arterial repair within 10 hours from the time of injury, but suc-
cessful repairs have been achieved even after 24 or more hours.
A. Emergency Measures:
1. Control of hemorrhage - If a tourniquet has been necessary,
an attempt should be made during the period of resuscitation
to replace it with an adequate pressure dressing. A pneu-
matic tourniquet should be avoided if possible during repair,
although it may be necessary during part of the procedure.
If the wound is large, the ends of the bleeding artery may
be directly secured by surgical clamps, taking care to oc-
clude the vessel close to the lacerated ends.
2. Preoperative care of an ischemic extremity - Shock should
be energetically treated with adequate blood replacement.
A prolonged surgical procedure should usually be deferred
until shock is controlled. Note: Do not use vasoconstrictor
drugs or local heat, and do not elevate the extremity.

Adequate immobilization in a well-padded splint is important. It is probably best to keep the limb at room temperature.

An x-ray of the injured area may be of value to determine the presence of fractures or foreign bodies.

B. Surgical Treatment:

1. Debridement of the wound should be carried out as described in Chapter 1, Trauma and Emergencies.

2. Arterial suture - Care should be taken to make sure that an artery has not been injured in more than one place and that the posterior wall has not been injured also.

   Suture repair of a lacerated artery is suitable only for small, cleanly lacerated injuries; contused, irregular arterial wounds should be treated by excising the damaged area and an 8-10 mm. segment of apparently normal vessel wall on both sides. An end-to-end anastomosis can be done if technically feasible; if not, a graft can be used.

   The concomitant vein should be preserved when possible; if it has been damaged, it may have to be ligated.

   Care should be taken to cover the repaired artery, using muscle tissue if possible. If the injury is in a joint area, that area should be splinted for 2-3 weeks.

   Bounding pulses may be noted in the distal arteries at the close of the procedure; occasionally pulses will not be noted for a few hours after the repair even though there is clinical evidence of capillary circulation. If on the other hand there is no evidence of return of circulation an hour or so after completion of a grafting procedure, the wound should probably be reexplored to determine if thrombosis has occured.

3. Arterial ligation - Ligation of a major artery in an extremity should be reserved for cases with arterial injury which will not tolerate the more prolonged surgery required for primary repair.

   a. A single artery in the lower arm or lower leg can be ligated; if two are injured, an attempt should be made to repair one of them even though the success rate in these small vessels is low.

   b. Back bleeding from the distal end of a divided artery does not give an accurate indication of adequacy of collateral circulation.

   c. In borderline cases, intermittent sympathetic blocks at six-hour intervals may be of importance in the early postoperative period. If block is effective, an emergency sympathectomy may be of value.

4. Associated fractures - Casts should be cut to allow ready access to the repaired vessel if secondary hemorrhage should result. Traction should be applied with extreme care so as not to disrupt the anastomosis.

5. A pulsating hematoma which does not increase progressively in size and is associated with a small or distant skin wound will go on to form a false or traumatic aneurysm; in this case, surgical correction of the defect is best deferred for weeks or months.

Prognosis.

Prognosis for survival of a limb which has sustained a severe arterial injury depends upon the following:

A. Duration of Impaired Oxygenation: In general, injuries which are repaired within 10 hours have the most favorable prognosis.

B. Management: Simple ligation of a major artery has a high subsequent amputation rate (50-75%), whereas the amputation rate in cases where arterial continuity has been restored is only about 10%.

C. Associated Injuries: If there is a fracture at or below the level of the arterial injury, the prognosis for survival of the limb is considerably worse.

TRAUMATIC ARTERIOVENOUS FISTULA

An arteriovenous fistula may result from a clean-cut knife wound or a through-and-through bullet wound which produces injury to both artery and vein but usually little associated damage to other structures. In most cases bleeding can be controlled by only slight pressure, and pulsating hematomas may be mild and temporary because the artery bleeds more easily into the vein than into the tissues. Since the artery is often only partially divided, gangrene may not be imminent; and if the circumstances do not demand immediate surgery there are several advantages to delaying surgery until the initial wound has healed and the tissue reaction has subsided: (1) The hazards of operating through contaminated tissue are avoided. (2) Surgery may be difficult because the anatomy is altered by the associated hemorrhage. (3) More adequate collateral circulation develops in a number of weeks. (4) Spontaneous closure of a fistula occasionally occurs.

However, if the injury is associated with a progressively expanding hematoma, emergency surgery may be needed in order to save the limb or the patient's life.

Clinical Findings.

In an unrepaired traumatic arteriovenous fistula a progressive dilatation of the vessels closely associated with the fistula occurs. Collateral circulation gradually develops around the lesion. The larger the fistula, the greater the amount of collateral circulation which develops, and the artery distal to the defect will often enlarge as a result of blood flowing into it from the enlarged collateral channels. If the fistula is large, the direction of blood flow in the artery just distal to the fistula will be toward the fistula and the low-pressure venous circulation beyond. Arteriosclerotic changes gradually occur in the proximal artery, in the fistula, and occasionally in the vein. A palpable thrill and an audible bruit are often present.

Complications.

A. Varicose veins and sometimes ulcers may develop in the extremity distal to the fistula, and considerable edema and fibrosis may be present secondary to the impaired venous return.

B. Cardiac Manifestations: Centrally located fistulas are more dangerous than a fistula of the same size in a more peripheral part of the vascular tree; this is because the volume of flow

through a fistula is greater in the centrally located lesions.

1. Tachycardia is present, which slows if the fistula is temporarily occluded (Branham's sign). The diastolic pressure is lowered by the persistent reduction of the peripheral resistance caused by the fistula, and an increased pulse pressure results. "Water hammer" pulse may be noted.
2. The total blood volume of the body gradually increases to compensate for the blood flow through the fistula.
3. Cardiac dilatation and, to a lesser extent, hypertrophy results from the increased venous return and cardiac output.
4. High output cardiac failure may result which can lead to death.

Treatment.

Repair of the defect should be carried out soon after the initial wound has healed and the tissue reaction has subsided in order to prevent local or cardiac complications.

The fistula should be excised and, if possible, a primary anastomosis carried out between the ends of the artery. An artery prosthesis should be used if any degree of tension is required to bring the vessel ends together. The vein is usually ligated above and below the fistula.

DEGENERATIVE AND INFLAMMATORY ARTERIAL DISEASES

ANEURYSMS OF THE THORACIC AND UPPER ABDOMINAL AORTA

Progress in antibiotic therapy has reduced the incidence of syphilitic aneurysms; most thoracic aneurysms are now arteriosclerotic in nature. Very rapid deceleration, as in an automobile or airplane accident, can result in a tear of the thoracic aorta just beyond the origin of the left subclavian artery. Cystic medial necrosis, a poorly understood and relatively rare degenerative condition, may lead to a thoracic aneurysm in relatively young people.

Clinical Findings.

Manifestations depend largely on the position of the aneurysm and its rate of growth.

A. Symptoms and Signs: There may be no symptoms, or there may be pain in the substernal, back, or neck areas, and pressure manifestations on (1) the trachea (dyspnea, stridor, a brassy cough), (2) the esophagus (dysphagia), (3) the left recurrent laryngeal nerve (hoarseness), or (4) the superior vena cava (edema in the neck and arms; distended neck veins). The findings of regurgitation at the aortic valve may be present.
B. Laboratory Findings: Serologic tests for syphilis may be positive.
C. X-ray Findings: X-ray examination (several views) and possibly fluoroscopy, tomography, esophagraphy, and angiocardiography are the chief means of arriving at a diagnosis.

Differential Diagnosis.

The differential diagnosis between an aneurysm and a mediastinal tumor can sometimes be made only at thoracotomy, and even then the diagnosis is often difficult since some aneurysms are so filled with clotted blood that pulsations may not be a prominent feature.

Treatment.

Aneurysms of the thoracic aorta usually progress, with increasing symptoms, and finally rupture. Resection of the aneurysm is now considered the treatment of choice.

A. Saccular Aneurysms With Narrow Necks: These can usually be removed at the neck without cross-clamping the aorta; the aorta can then be closed with a series of sutures at the junction of the neck of the sac with the aorta.

B. Larger Saccular and Fusiform Aneurysms: These require resection of the aneurysm with an aortic graft to the defect. Temporary occlusion of the aorta to interrupt the blood flow to the spinal cord, kidneys, and sometimes the brain is necessary. Among the technics which have been used to maintain circulation to vital areas during the period of occlusion are (1) extracorporeal circulation by means of a pump oxygenator (for aneurysms of the ascending aorta); (2) a temporary shunt from the aorta above to the aorta below the aneurysm; and (3) pumping of blood from the left atrium to the femoral artery (for aneurysms located along the aorta from the arch to beyond the renal arteries). Hypothermia reduces the metabolic requirements of the spinal cord and brain and thus prolongs the safe period of interrupted aortic circulation for a number of minutes.

DISSECTING ANEURYSMS OF THE AORTA

The dissection may begin as a primary tear of the intima of an atherosclerotic ascending aorta or, less commonly, the arch or distal aorta. Hypertension often is an etiologic factor. Dissection may arise as a result of degenerative changes in the media of the aorta and intramural hemorrhage, followed later by a tear of the intima. In either case, the blood dissects in the media of the artery, and external rupture usually occurs in hours, days, or weeks. Intramural rupture may occur, and the patient may survive with blood flowing through both the normal and the new channel.

Clinical Findings.

A. Symptoms and Signs: Severe, persistent pain of sudden onset is usually present in the chest and occasionally radiates to the back, abdomen, and hips. Shock may be present. Partial or complete occlusion of the arteries to the brain or spinal cord may lead to CNS findings. Peripheral pulses and blood pressures may be diminished and unequal.

B. X-ray Findings: Roentgenograms may reveal widening of the thoracic aorta with progressive changes. The double lumen can sometimes be demonstrated by angiocardiography.

Treatment.
The objective of surgical treatment is to prevent further intra-mural dissection and eventual rupture by creating a reentry from the dissecting, intramural channel to the true lumen of the aorta. A shunting procedure or hypothermia is usually necessary to pro-tect the spinal cord while the thoracic aorta is clamped.

Prognosis.
The mortality in unoperated cases is quite high: death usually occurs in a matter of days or weeks unless surgical treatment is prompt and adequate.

ABDOMINAL AORTIC ANEURYSMS

The vast majority of aneurysms of the abdominal aorta are be-low the origin of the renal arteries and often involve the common iliac arteries. Aneurysms of the upper abdominal aorta are rare. Most aneurysms of the distal aorta are arteriosclerotic in origin and fusiform in shape.

Clinical Findings.
  A. Symptoms and Signs: Three phases in the medical course can be recognized:
    1. Asymptomatic - A pulsating mid and upper abdominal mass may be discovered on a routine physical examination, most frequently in men over 50 years of age. As a general rule, surgical resection should be advised even for asymptomatic aneurysms, particularly if the aneurysm is large. Although small aneurysms (less than 7 mm.) also rupture, surgery may occasionally be withheld if the patient is a poor opera-tive risk and especially if he has significant cardiac, renal, or distal peripheral obliterative vascular disease. If the aneurysm increases in size or becomes symptomatic, the decision not to operate should be reviewed.
    2. Symptomatic - Pain varies from mild mid-abdominal dis-comfort to severe constant or intermittent abdominal and back pain requiring narcotics for relief. Intermittent pain may be associated with a phase of enlargement or intra-mural dissection. Pain is an unfavorable prognostic sign which usually justifies early surgery. Peripheral emboli and thrombosis, which commonly complicate the more distal aneurysms, are infrequently present in abdominal lesions.
    3. Ruptured - Rupture of an aneurysm almost always causes death in a few hours or days and is therefore an indication for immediate surgical removal. Pain is usually severe. Because the dissection is most often into the retroperitoneal tissues, which offer some resistance, shock and other mani-festations of blood loss may at first be mild or absent; but free uncontrolled bleeding inevitably follows, resulting in death. There is an expanding, pulsating abdominal and flank mass, and subcutaneous ecchymosis is occasionally present in the flank or groin. Massive gastrointestinal hemorrhage from the aneurysm through a fistula in the third portion of the duodenum can occur. About half of these

patients can be saved by immediate surgery.

B. Laboratory Findings: Cardiac and renal function should be evaluated by means of Ecg., urinalysis, and BUN determination.

C. X-ray Findings: Calcification in the wall of the aneurysm will usually outline the lesion on anteroposterior and lateral plain films of the abdomen and intravenous urograms. In some cases the position of the aneurysm in relation to the renal arteries can be determined. Bony erosion of the vertebrae does not usually occur in abdominal aneurysms. Translumbar aorto-grams are seldom employed; the aneurysm may be ruptured by the procedure, and the information gained regarding the upper limits of the lesion may be inaccurate since a thrombus often occupies most of the aneurysm. If the amount of distal arterial occlusive disease must be determined, an aortogram may be indicated.

Treatment.

Surgical excision and grafting of the defect is indicated on all aneurysms of the distal aorta except when the lesion is very small and asymptomatic or when the general condition of the patient does not permit surgery.

Prognosis.

Complications and mortalities following surgery are most often associated with myocardial infarction, acute renal failure, hemor-rhage from one of the anastomoses, and distal arterial thrombosis. The mortality rate following elective surgical treatment is 5-15%. When rupture has occurred, surgical mortality is 50%.

ANEURYSMS OF THE VISCERAL BRANCHES OF THE ABDOMINAL AORTA

The hepatic, splenic, and more rarely the renal and superior mesenteric arteries occasionally are subject to aneurysmal changes. Treatment is by resection with or without reestablishment of circu-lation depending upon the nature of the aneurysm and the importance of the vessel.

FEMORAL AND POPLITEAL ANEURYSMS

Aneurysms of the femoral or popliteal arteries are not uncom-mon. They are usually arteriosclerotic in origin, in which case they may be multiple and are often bilateral. They may also be due to trauma. Syphilitic or mycotic aneurysms occur occasionally, the latter after an episode of bacteremia.

Clinical Findings.

The diagnosis is usually not difficult, although a lesion in the popliteal area may go unnoticed until attention is focussed on the area by a complication.

A. Symptoms and Signs: The cardinal finding is a firm, pulsating mass in the femoral or popliteal area, often associated with a bruit. Pulsation may not be present if thrombosis has already

taken place. The distal circulation may be impaired, espe-
cially if the gelatinous thrombus which so frequently fills most
of the aneurysm has partially or completely occluded the cen-
tral lumen or if emboli from this thrombus have blocked 1 or
more of the distal vessels.
B. X-ray Findings: X-rays with arteriography may be helpful in
outlining the extent of the aneurysm and the status of the periph-
eral vessels, although the central getatinous thrombus which
is so often present may make interpretation difficult.

Complications.
Rupture can occur, and often results in death or the loss of the
limb. Complete thrombosis causes distal gangrene in about one-
third of popliteal aneurysms. Emboli to the distal vessels from
the thrombus lining the wall of the aneurysm often cause ischemic
changes or gangrene. Thrombophlebitis can occur as a result of
pressure obstruction of a neighboring vein. Pressure on the tibial
or peroneal nerves may produce pain in the lower leg.

Treatment and Prognosis.
Surgical excision of the aneurysm with grafting of the defect is
the treatment of choice in all except very small femoral or popliteal
aneurysms or in patients who are poor surgical risks. The inci-
dence of loss of limb following resection and grafting is low. Throm-
bophlebitis is a not infrequent postoperative complication even though
care is taken to avoid injuring or dividing the neighboring veins. In
the popliteal area, venous ligation may be necessary.

CAROTID ARTERY ANEURYSMS

Aneurysms of the carotid artery are relatively rare. The bi-
furcation of the common carotid is often involved. Pressure symp-
toms and pain are sometimes present, and a pulsating mass is
readily seen and felt. The complications include rupture, throm-
bosis, or emboli from the aneurysm to the cerebral vessels.

Treatment.
Small fusiform aneurysms may be wrapped with strips of fascia
lata. Larger aneurysms must be resected, and a graft is often re-
quired to bridge the defect. Since surgery may be complicated by
CNS damage, hypothermia or an internal or external shunt should
be employed during the period of occlusion.

OCCLUSIVE DISEASE OF THE AORTA   •
AND ILIAC ARTERIES

Occlusion of the aorta and the iliac arteries begins most fre-
quently just proximal to the bifurcation of the common iliac arteries
as atherosclerotic changes in the intima and media, often with as-
sociated perivascular inflammation and calcified plaques in the
media. Progression is usually in a proximal direction, involving
first the aortic bifurcation and then the abdominal aorta (particularly
below the renal vessels). Narrowing progresses to complete throm-

botic occlusion, which often extends from just below the renal arteries to the distal common iliac arteries. Although atherosclerosis is a generalized disease, occlusion tends to be segmental in distribution, and when the involvement is in the aortic iliac vessels there is often minimal atherosclerosis in the more distal external iliac and femoral arteries. The best candidates for direct arterial surgery are those with localized occlusions with relatively normal vessels above and below.

Men between the ages of 30 and 60 are most commonly affected. Cardiac and renal diseases and hypertension are often associated with this defect.

Clinical Findings.

A. Symptoms and Signs: Intermittent claudication is almost always present in the calf muscles and is usually present in the thighs and hips also. It is most often bilateral and progressive, so that by the time the patient seeks help the pain may be produced by walking a block or less. Difficulty in sustaining an erection is a common complaint in men. Coldness of the feet may be present; rest pain is infrequent.

Femoral pulses are absent or weak. Pulses distal to the femoral area are usually absent. Pulsation of the abdominal aorta may be palpable. A palpable thrill or bruit over an artery is usually indicative of narrowing of the lumen at that site or proximally. Atrophic changes of the skin, subcutaneous tissues, and muscles are usually minimal or absent. Dependent rubor and coolness of the skin of the foot are minimal.

B. Laboratory Findings: Cardiac and renal function should be evaluated preoperatively to determine the degree of coronary and degenerative renal disease.

C. X-ray Findings: A translumbar aortogram gives much valuable information regarding the level and extent of the occlusion and the condition of the vessels distal to the block. However, this procedure is dangerous and the possibility of a complication must be weighed against the importance of the information to be gained. Anteroposterior and lateral films of the abdomen and sometimes of the thighs will give some information regarding the degree of calcification in the vessels. An intravenous urogram is often of value.

Treatment.

Surgical treatment is indicated if claudication interferes appreciably with the patient's activities or if previously minimal symptoms show signs of progression. The objective of treatment is reestablishment of blood flow through the narrowed or occluded aorto-iliac segment. This can be achieved by arterial prosthesis or thrombo-endarterectomy.

A. Arterial Graft (Prosthesis): Arterial prosthesis, replacing or bypassing the occluded segment, is probably the treatment of choice in the more extensive aorto-iliac occlusions.

B. Thrombo-endarterectomy: This is perhaps of most value when the occlusion involves only a short segment of artery.

C. Sympathectomy: A bilateral lumbar sympathectomy may be added to the direct arterial procedure.

Complications of Treatment.

The complications most frequently encountered are (1) hemorrhage from the suture line, (2) thrombosis of the prosthesis or the endarterectomized artery or of vessels in the legs, (3) postoperative oliguria or uremia, and (4) myocardial infarction from pre-existing coronary artery disease.

Prognosis.

The operative mortality is relatively low and the immediate and long-term benefits are usually marked. Improvement is both subjective and objective, with relief of all or most of the claudication and in many cases return of all of the pulses in the legs.

OCCLUSIVE DISEASE OF THE FEMORAL AND POPLITEAL ARTERIES

In the region of the thigh and knee the vessels most frequently blocked by occlusive disease are the superficial femoral artery and the popliteal artery. Atherosclerotic changes usually appear first at the most distal point of the superficial femoral artery where it passes through the adductor magnus tendon into the popliteal space. In time the whole superficial femoral artery and the proximal popliteal artery may become occluded, but often the common femoral and profunda femoral arteries are patent and relatively free from disease. The status of the popliteal artery and its 3 main branches (the anterior and posterior tibial arteries and the common peroneal artery) is important from the point of view of direct surgical therapy since thrombosis will occur if moderate or marked atherosclerosis in this area prevents an adequate distal "run-off" of blood.

Clinical Findings.

As a rule the changes are initially more advanced in 1 leg than the other, although similar changes often appear later in the other extremity.

A. Symptoms and Signs: Intermittent claudication, which often appears upon as little exertion as walking one-half to 1 block, is confined to the calf and foot. If rest pain is also present, arterial disease is extensive and the prognosis is poor. Atrophic changes in the lower leg and foot may be quite definite, with loss of hair, thinning of the skin and subcutaneous tissues, and diminution in the size of the muscles.

Dependent rubor and blanching on elevation of the foot is usually present. When the leg is placed in a dependent position after being elevated, venous filling on the dorsal aspect of the foot may be slowed to 15 or 20 seconds. The foot is usually cool or cold.

The common femoral pulsations are usually of fair or good quality, but no popliteal or pedal pulses can be felt.

B. X-ray Findings: Roentgenograms of the thigh and leg may reveal calcification of the superficial femoral and popliteal vessels. A femoral arteriogram will show the location and extent of the block as well as the status of the distal vessels.

Treatment.
    Surgery is indicated (1) if intermittent claudication interferes
significantly with the patient's activities or (2) if pregangrenous or
gangrenous lesions appear on the foot and it is hoped that a major
amputation can be avoided.
  A. Arterial Graft (Prosthesis): An arterial prosthesis can be
     placed around the occluded segment, using the bypass technic
     with end-to-side anastomoses both proximally and distally.
  B. Thrombo-endarterectomy: Thrombo-endarterectomy with re-
     moval of the central occluding core can be carried out by
     means of specially designed instruments which dissect in the
     cleavage plane in the wall of the diseased artery. Part of the
     media and the adventitia is left.
  C. Sympathectomy: Lumbar sympathectomy may be used as an
     adjunct to grafting or endarterectomy or as the sole measure
     if a direct operation is thought inadvisable. It can be done a
     few days before or at the same time as a direct procedure;
     the vasodilator effect of sympathectomy may improve the cir-
     culation to the foot and lower leg.

Prognosis.
    Thrombosis of the "bypass" grafts or of the endarterectomized
vessels either in the immediate postoperative period or months or
years later is relatively frequent. However, this is usually no more
disabling than the occlusive disease, and if the procedure is success-
ful the extremity may be considerably improved.

OCCLUSIVE DISEASE OF THE ARTERIES IN THE
LOWER LEG AND FOOT

    Occlusive processes in the lower leg and foot may involve, in
order of incidence, the tibial and common peroneal arteries and
their branches to the muscles, the pedal vessels, and occasionally
the small digital vessels. Symptoms depend upon the vessels that
are narrowed or thrombosed, the suddenness and extent of the oc-
clusion, and the status of the proximal and collateral vessels. The
clinical picture may thus vary from slowly developing vascular in-
sufficiency coming on over months or years and resulting ultimately
in atrophy of the skin, ischemic pain, and finally gangrene, to a
rapidly progressive and extensive thrombosis resulting in acute
ischemia and often gangrene.

Clinical Findings.
    Although all of the possible manifestations of vascular disease
in the lower leg and foot cannot be described here, there are certain
significant clinical aspects which enter into the evaluation of these
patients.
  A. Symptoms:
     1. Claudication - Intermittent claudication is the commonest
        presenting symptom. Aching fatigue during exertion usually
        appears first in the calf muscles; in more severe cases a
        constant or cramping pain may be brought on by walking only
        a short distance. Less commonly the feet are the site of
        most of the pain. The distance that the patient can walk

before pain becomes severe enough to necessitate a few minutes' rest gives a rough estimate of circulatory inadequacy: 2 blocks (400-500 yards) or more is mild, 1 block is moderate, and half a block or less is severe.

2. Rest pain - Rest pain may be due to sepsis or simple ischemia. The former is usually throbbing, whereas ischemia usually produces a persistent, gnawing ache with occasional spasms of sharp pain. Rest pain first comes on in bed when the foot is warm, and some relief is obtained by uncovering the foot and placing it in a dependent position. In more advanced stages the pain may be constant and so severe that even narcotics may not relieve it. The patient may request amputation.

B. Signs:

1. Absence of pulsations - Careful palpation over the femoral, popliteal, dorsalis pedis, and posterior tibial arteries should be done to determine if pulsations are present. Although the popliteal pulse may be present, both pedal pulses are usually absent. The dorsalis pedis pulse is congenitally absent in 8% of people, but the posterior tibial pulse is always present unless the artery is diseased.

Claudication and atrophic changes can occur in patients with palpable pedal pulses. If the popliteal pulse is present, a direct surgical approach on the vessels of the leg is not likely to be of any value.

2. Color changes in the feet - Normally the skin of the feet is warm and pale as a result of the rapid flow of blood through the arterioles, and few of the capillaries are filled. Skin irritation leads to capillary filling and the skin becomes red. Defective blood supply causes anoxic paralysis of the capillaries and a bluish-red skin.

The rate of return of color following blanching induced by local pressure is an inaccurate index to circulatory adequacy because the blood which returns on release of the pressure does not necessarily represent true circulation.

a. Pallor upon elevation - If pallor appears rapidly upon elevation of the foot from the horizontal - or if it appears when the leg is only slightly raised, the circulatory status is poor.

b. Flushing time - Color normally returns in a few seconds to a foot placed in a dependent position after 1-2 minutes of elevation. The poorer the collateral circulation, the longer the interval before flushing begins to appear in the toes. In general, if dependent flushing begins in the toes within 20 seconds, the collateral circulation is probably adequate and the extremity may be salvaged by lumbar sympathectomy. If flushing time is over 20 seconds, the arterial disease is extensive and sympathectomy will be of little or no value.

c. Dependent rubor - Beefy redness of the toes or foot on dependency is frequently present in occlusive disease of this area, but it may not reach its full extent until a minute or more after the leg has been placed in the dependent position. Dependent rubor represents ischemic paralysis of the capillaries in the skin, and implies

moderate or severe arterial occlusive disease.

d. Rubor of stasis - When almost complete stasis in the distal vessels occurs, with venous as well as arterial thrombosis and extravasation of red blood cells, redness of the toes and forefoot may develop which may not completely disappear on elevation of the leg. This disorder is often associated with severe pain and is more commonly noted in thromboangiitis obliterans than in atherosclerosis.

e. Patchy cyanosis and pallor indicates a severe degree of ischemia; it is seen frequently following acute thrombosis or recent embolus.

3. Venous filling time - If the valves in the saphenous system are competent, venous filling is a valuable gauge of collateral circulation of the foot. If the veins on the dorsum of the foot begin to fill in 30 seconds or less after the leg is placed in a dependent position after having been elevated for about 1 minute, the borderline pregangrenous extremity can probably be saved by sympathectomy.

4. Local tissue changes - Diminished arterial flow causes wasting of the subcutaneous tissues of the digits, foot, and lower leg. Hair is lost over these areas, the skin becomes smooth and shiny, and the nails become thickened and deformed. Infections are common following minor injuries or even without injury at the edge of a nail or under a thick callus. Once established, infection may become indolent and chronic or may lead to localized or progressive gangrene. Local heat should not be used in the treatment of such an infection.

5. Skin temperature studies - Skin temperature and plethysmographic studies may be of value in calibrating the degree of vasoconstrictor activity, and in certain borderline cases may help determine whether lumbar sympathectomy will be of value. A rough clinical test may be carried out by exposing the leg to room temperature for a few minutes. If arterial circulation is inadequate the leg will feel quite cool, especially if there is considerable vasoconstrictor activity or if the leg is quite ischemic.

6. Sweating - Sweating is under the control of the sympathetic nervous system and is therefore an index to the degree of autonomic activity in the extremity. If a patient with occlusive disease still notes sweating of his feet, some degree of sympathetic activity is present and lumbar sympathectomy is likely to be of benefit.

C. X-ray Findings: Films of the lower leg and foot may show calcification of the vessels and thinning of the bones. If there is a draining sinus or an ulcer close to a bone or joint, osteomyelitis may be apparent on the film. If fairly strong popliteal pulses can be felt, arteriography is not likely to be of much value as a guide to surgical treatment; sometimes, however, the status of the femoral or popliteal arteries must be evaluated in this way.

Complications in Diabetic Patients.

Atherosclerosis develops oftener and earlier in patients with diabetes mellitus, especially if the disease has been poorly controlled over a period of years. The care of the diabetic patient who develops occlusive arterial disease of the foot and lower leg is more difficult. The resistance to infection seems to be less, and the control of diabetes may be more difficult in the presence of infection. Anemia and hypoproteinemia are not infrequently present in moderate or severe diabetes and tend to be resistant to therapy; tissue ischemia may be made more severe by this anemia, and blood transfusions may be necessary. Anesthesia of the toes and distal foot (due to diabetic neuropathy) predisposes to injury and secondary infection. Visual defects due to diabetic retinopathy make care of the feet more difficult and injuries more likely.

Treatment.

A. Intermittent Claudication: The patient should be instructed to walk slowly, take short steps, avoid stairs and hills, and to stop for brief rests to avoid severe pain.

Lumbar sympathectomy is the surgical treatment of choice. It is most likely to be of benefit if the popliteal pulse is palpable, but it may still be of value when only the femoral pulse can be felt in the leg.

A flushing time of 20 seconds or less when the leg, blanched by a period of elevation, is placed in the dependent position is a favorable sign; if it develops after 30 seconds, sympathectomy will probably be of no benefit. If sweating of the feet occurs, the operative result is likely to be favorable. More refined objective evidence for or against the operation may be obtained in constant temperature rooms and by means of the plethysmograph.

Walking capacity following sympathectomy may never improve or may not improve for weeks or months since the increased collateral flow to the muscles develops gradually. The improvement in skin circulation, with a dry, warm foot, is often apparent within a few hours.

Because bilateral sympathectomy may result in considerable paralytic ileus in older patients, 2 procedures with 5-7 days between is probably more satisfactory and can be done with a low mortality.

A frequent and annoying sequel to sympathectomy is an unexplained neuralgia down the lateral aspect of the thigh and leg which develops usually by about the tenth postoperative day. The pain may be mild or severe, and may last for days or weeks. The treatment is symptomatic, and the neuralgia always ultimately disappears.

B. Circulatory Insufficiency in the Foot and Toes: Sympathectomy is often indicated even when the femoral pulse is absent or when pregangrenous changes are present in the toes.

Moderate or marked rest pain usually implies such advanced changes that the procedure will be of little or no benefit. The operation usually results in a dry, warmer foot, and the additional collateral flow serves to protect the leg against future vascular occlusions.

C. Infections, Ulcers, and Gangrene of the Toes or Foot:

1. Early treatment of acute infections - Place the patient at complete bed rest with the leg in a horizontal or slightly depressed position. An open or discharging lesion should be covered with a light gauze dressing, but tape should not be used on the skin. Culture and sensitivity studies should be obtained if there is any purulent discharge, but if advancing infection is present an appropriate antibiotic should be started immediately. Purulent pockets should be gently drained.

Ulcerations covered with necrotic tissue can often be prepared for spontaneous healing or grafting with wet dressings of sterile saline changed 2-3 times a day.

Control diabetes and anemia, if present.

2. Early management of established gangrene - In most instances an area of gangrene will progress to a point where the circulation provided by the inflammatory reaction is sufficient to prevent progressive tissue death. The process will at least temporarily demarcate at that level. This can be encouraged by measures similar to those outlined in the preceding section on the treatment of acute infection. If the skin is intact and the gangrene is dry and due only to arterial occlusion, antibiotics should be withheld. If infection is present or if the gangrene is moist, antibiotics should be used in an effort to limit the process and prevent septicemia.

If the gangrene involves only a segment of skin and the underlying superficial tissue, sympathectomy and, if possible, an artery graft may reverse the process. The necrotic tissue can be removed and the ulcer grafted or allowed to heal as outlined above in the section on ulcers. Occasionally, if a toe is involved, a lower amputation can be carried out by means of a more aggressive approach.

3. Amputations for gangrene -

   a. A toe which is gangrenous to its base can sometimes be amputated through the necrotic tissue and left open; this procedure may be employed to establish adequate drainage when there is active infection with undrained pus in addition to the gangrene.

   b. When the distal part of the toe is gangrenous and there is sufficient circulation in the proximal toe, a closed amputation can be carried out after the area has become well demarcated and inflammation has subsided.

   c. Transmetatarsal amputation can be considered if the gangrene involves 1 or more toes down to but not into the foot and if the circulation in the distal foot seems adequate to support healing.

   d. Amputation below the knee may be employed in patients with a palpable popliteal pulse or good collateral circulation around the knee (as indicated by a warm and well-nourished lower leg) when gangrene or ischemia in the foot is so severe or so distributed that local amputation is not feasible.

   e. Amputation above the knee (through the distal thigh in the supracondylar area) is indicated in patients with advanced peripheral vascular disease requiring amputation because

of gangrene or severe ischemic pain. Even if the femoral artery is obliterated, there will be sufficient collateral circulation to allow healing provided gentle technic with good hemostasis is used.

f. Guillotine amputation - Infection with bacteremia or septicemia occasionally develops secondary to gangrene of the lower extremity. This can usually be controlled only by emergency amputation above or below the knee. In such a situation, it is often wise to leave the stump open so that it can heal by secondary intention or be revised or reamputated when the infection has been controlled.

Prophylaxis of the Complications of Impaired Circulation.
A. General Measures:
1. Care and control of cardiac disease, diabetes mellitus, anemia, hypoproteinemia, and obesity.
2. Tobacco should be prohibited.
3. Undue exertion and fatigue are not desirable.
4. Alcoholic beverages in moderation may be of value.
B. Local Measures:
1. The patient should be instructed about the dangers of foot infections and injuries; if sensation is altered, the significance and dangers of this should be discussed. Warn the patient that he should consult a physician promptly even in the event of minor injuries or infections.
2. Lanolin should be rubbed on the skin each night to keep it pliable and free of fissures and thick calluses; the nails should be trimmed straight across and not too short in order to prevent ingrown nails; thick calluses and corns should be trimmed by a chiropodist or physician; and extremes of temperature should be avoided.
3. Socks (preferably soft wool) should be changed once a day. Shoes should be well fitted, and great care should be taken in breaking in new shoes. The patient should never walk barefoot.

OCCLUSIVE DISEASE OF THE INNOMINATE, CAROTID, VERTEBRAL, AND SUBCLAVIAN ARTERIES

Occlusion of 1 or more of these arteries can lead to signs and symptoms referable to the CNS or in the eyes or arms. Narrowing or occlusion is usually at or just beyond the bifurcation of the common carotid artery. A significant number of patients with arterial insufficiency involving the brain and arms have segmented occlusive processes which are amenable to surgical correction.

The aortic arch syndrome may be due to syphilitic aortitis with or without aneurysm, atherosclerotic occlusive disease, or a nonspecific occlusive arteritis of the major aortic branches in young women. Narrowing or occlusion of the carotid artery beyond its origin is almost always atherosclerotic.

Clinical Findings.
A. Symptoms and Signs:
1. CNS - Common manifestations are vertigo (especially on

standing up), unsteady gait, fainting spells, memory and
personality changes, speech disorders, headaches, hemi-
paresis or hemiplegia, absent or weak common carotid
pulsations on 1 or both sides, and a systolic murmur at the
angle of the jaw. If obstruction is partial, these changes
may be transient and episodic and probably are related to
variations in collateral circulation and BP. The manifes-
tations are usually predominantly one-sided and slowly
progressive.

2. Eye - Transient episodes of altered vision or blindness
(especially when walking), rapidly developing cataracts,
changes in the fundus (e.g., optic nerve atrophy or retinal
arteriovenous anastomoses), and decreased intraoptic ar-
terial pressures (as measured by ophthalmodynamometry).

3. Arms and face - Intermittent claudication involving the arm
or the muscles of mastication; weakness or atrophy of the
arm or facial muscles; absent subclavian, brachial, and
radial pulsations; diminished or absent BP in the arm, with
normal or elevated BP in the lower extremity and a systolic
murmur in the supraclavicular area.

B. X-ray Findings: Angiography by means of a retrograde aorto-
gram with the catheter introduced through a femoral artery, an
angiocardiogram, or a percutaneous or a cut-down carotid
arteriogram may show the position and degree of the occlusive
process.

Treatment.
A. Indications: The decision to operate depends upon the benefits
which may be expected following correction of the deficiency of
the blood supply to the brain.

1. Emergency surgery - Serious consideration should be given
to emergency evaluation and operation in patients who sud-
denly develop major neurologic changes that could be due
to a partial or complete occlusion of the carotid or vertebral
vessels.

2. Elective surgery - Patients with intermittent symptoms or
constant mild neurologic defects - or those who have
recovered well from a major vascular insult - should be
evaluated for surgery.

3. Withhold surgery - Patients with complete hemiplegia who
show no signs of recovery will probably not be benefited by
an operation to improve the blood supply to the damaged
brain. If the occlusion of the internal carotid artery is
complete and is more than a few hours old, the vessel will
probably be thrombosed beyond the cervical segment of the
internal carotid artery and damage to the brain will be ir-
reversible.

B. Surgical Treatment:
1. Short or localized partial or complete occlusion - The best
treatment is by thrombo-endarterectomy of the involved
segment, which is usually located either at or just beyond
the aortic arch or, more commonly, at the junction of the
common carotid artery. Occlusion of the proximal 1-2 cm.
of the vertebral artery is best approached by opening the

subclavian artery opposite the origin of the occluded verte-
bral vessel.

2. Longer, segmental occlusions (especially those close to the
arch of the aorta) - These are usually treated by a bypass
type of arterial graft with an end-to-side anastomosis be-
tween the graft and the ascending aorta.

Prognosis.

The results are better when the occlusions are close to the arch
of the aorta, and are less satisfactory when the internal carotid or
vertebral vessels are involved. Complete occlusion of the distal
carotid artery and continuous and marked CNS changes imply a poor
prognosis for surgical therapy, whereas the results are better when
there are only transient episodes of cerebral dysfunction without
significant neurologic defects and when a point of localizing obstruc-
tion can be demonstrated.

RENAL ARTERY STENOSIS*

Stenosis of a renal artery can cause progressive hypertension
which may be relieved if an unobstructed blood flow is restored to
the kidney. Atherosclerosis is the commonest cause, but congenital
stenosis of the renal artery does occur and may account for hyper-
tension on this basis in children or young adults.

Clinical Findings.

A. Symptoms and Signs: Renal artery stenosis should be con-
sidered in any patient with rapidly developing, severe hyper-
tension, especially if the diastolic pressure is high or if a bruit
can be heard over the area of a renal artery, and in the follow-
ing types of cases:

1. Hypertension which has its onset either in old people or in
those under 30 years of age.

2. Hypertension which develops or progresses after an episode
of flank or abdominal pain.

3. Hypertension in patients in whom intravenous urograms re-
veal a definite difference in the size of the kidneys or in the
time taken for the first sign of the contrast media to appear
in each renal pelvis (frequent films in the first 10 minutes
may be necessary to determine the degree of difference).

B. Laboratory and X-ray Findings:

1. X-ray findings - Although a difference between the 2 kidneys
may be noted as mentioned above, this is frequently not the
case. Aortography with the tip of the needle or catheter
placed just above the orifices of the renal artery and using
a small amount of contrast media may reveal stenosis of 1

*The abdominal aorta and the common iliac artery are the vessels
which most frequently become atherosclerotic; however, the main
visceral branches may become involved in 1 or more of the following
ways: (1) thickening of the intima, which narrows the aortic orifice;
(2) a concentric area of thickening just inside the orifice; and (3)
plaque formation inside the orifice involving only part of the circum-
ference.

of the renal arteries. This is probably the most valuable single study and certainly should be carried out in any patient in whom the diagnosis of renal artery stenosis is seriously entertained. Angiocardiograms, using a large amount of contrast media, careful timing, and multiple films so as to catch the dye as it reaches the renal arteries is perhaps a safer although less exact way of demonstrating stenosis; a relative hypotension at the time of the study induced by medical means may be of value.

2. Laboratory findings - Differential urinary excretion studies of sodium, water, and PSP by means of catheters passed to each renal pelvis are of value provided the collection of samples from each renal pelvis is accurate.

If the above clinical studies point to the diagnosis of a renal artery stenosis, the accuracy of the diagnosis can be tested by differential arterial pressure studies at the time of surgical exploration; if a true stenosis exists, the aortic pressure is at least 30-40 mm. Hg higher than the pressure in the distal part of the renal artery.

Treatment.

The treatment of choice is removal of the stenotic area; if this cannot be done, nephrectomy should be performed provided the other kidney is free from disease. The direct procedure on the artery may involve (1) resection of a short stenotic segment, with end-to-end anastomosis; (2) endarterectomy of an atherosclerotic area; (3) anastomosis of the splenic artery to the renal artery beyond the stenotic area (if the defect is in the left side); or (4) an artery graft.

If the diagnosis is accurate and surgical correction of the defect is successful, a dramatic drop in the hypertension results in a high percentage of cases.

CELIAC AND SUPERIOR MESENTERIC ARTERY DISEASE*

Although slight narrowing of the orifices of these vessels is rather common, sufficient occlusion to produce symptoms is relatively rare.

Clinical Findings. †
A. Symptoms and Signs: Postprandial pain (abdominal angina) and malabsorption of food without demonstrable gastrointestinal lesions are the principal manifestations. Vascular insufficiency in other areas reinforces the diagnosis. A systolic murmur above and to the left of the umbilicus may result from superior mesenteric artery stenosis.
B. X-ray Findings: Arteriography of the individual visceral arteries may demonstrate the lesion.
C. Exploratory Laparotomy: An essential part of certain exploratory operations in older patients is palpation of the pulsations of the major abdominal arteries. The arterial pressure

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*See note (*) on p. 423.
†The manifestations of acute thrombosis of the superior mesenteric artery are discussed on p. 273.

measurements in the visceral vessels distal to the stenosis may be one-fourth or less of the aortic pressure.

Treatment.

Thrombo-endarterectomy of the occluding atherosclerotic lesions at the origin and proximal part of the superior mesenteric artery or the celiac artery is the usual approach to this problem at the present time, although in some cases reimplantation of the superior mesenteric artery lower in the aorta (or a graft) may be indicated.

THROMBOANGIITIS OBLITERANS
(Buerger's Disease)

Buerger's disease is an episodic and segmental inflammatory and thrombotic process of the arteries and veins, principally in the limbs. It is seen most commonly in men between 25 and 40 years of age. The effects of the disease are almost solely due to occlusion of the arteries. The symptoms are primarily due to ischemia, complicated in the later stages by infection and tissue necrosis. The inflammatory process is intermittent, with quiescent periods lasting weeks or years.

The differential diagnosis between Buerger's disease and atherosclerotic peripheral vascular disease may be difficult or impossible. The differentiation is sometimes based on the age at which symptoms first appeared. However, atherosclerosis can appear in young adults, and the diagnosis of thromboangiitis obliterans is made less frequently now than was formerly the case.

The arteries of the legs are most commonly affected. The plantar and digital vessels and those in the lower leg (especially the posterior tibial artery) are most frequently involved. Occlusion of the femoral-popliteal arteries does not often occur. In the upper extremity, the distal arteries are most commonly affected. Different arterial segments may become occluded in successive episodes; a certain amount of recanalization occurs during quiescent periods.

Superficial migratory thrombophlebitis is a common early indication of the disease.

The etiology is not known, but a history of smoking is almost always obtained and little or no progress can be made in treatment if the patient continues to smoke.

Clinical Findings.

The signs and symptoms are primarily those of arterial insufficiency, and the differentiation from arteriosclerotic peripheral vascular disease may thus be difficult. Although the 2 diseases are similar in many ways, the following findings suggest Buerger's disease:

A. The patient is a man between 20 and 40 years of age who smokes.

B. There is a history of migratory superficial segmental thrombophlebitis, usually in the saphenous tributaries rather than the main vessel. A biopsy of such a vein will often give microscopic proof of Buerger's disease.

C. Rest pain is persistent and gnawing or aching, often interfering with sleeping and eating. This pain tends to be more pronounced than in the patient with atherosclerosis. Numbness, diminished sensation, and pricking and burning pains may be present.

D. The color of the digits and the distal foot may remain relatively unchanged by posture; the skin may not blanch on elevation, and will darken with dependency to the degree usually seen in the atherosclerotic group. The distal vascular changes are often asymmetric, so that not all of the toes are affected to the same degree.

E. Trophic changes are present, with painful indolent ulcerations along the nail margins.

F. There is usually evidence of disease in both legs and possibly also in the hands and lower arms. There may be a history or findings of Raynaud's phenomenon in the fingers or distal foot.

G. The course is usually intermittent, with acute and often dramatic episodes followed by rather definite remissions. When the collateral vessels as well as the main channels have been occluded, an exacerbation is more likely to lead to gangrene and amputation. The course in the patient with atherosclerosis tends to be less dramatic and more persistent.

Treatment.

The principles of therapy are the same as outlined for atherosclerotic peripheral vascular disease; but the long-range outlook is better in patients with Buerger's disease, so that when possible the approach should be more conservative and tissue loss kept to a minimum.

A. General Measures: Smoking must be given up; the physician should be emphatic and insistent on this point. The disease is almost sure to progress if this advice is not heeded.

B. Surgical Treatment:

1. Sympathectomy is useful in eliminating the vasospastic manifestations of the disease and aiding in the establishment of collateral circulation during the acute phase. It is also helpful in relieving the milder or moderate forms of intermittent claudication and rest pain and may aid in healing following amputation of a toe.

2. Arterial grafts - If the femoral pulse is present and the popliteal pulse is absent, a femoral arteriogram should be taken to assess the feasibility of using a graft. However, arterial grafting procedures are not often possible in patients with Buerger's disease because they do not usually have a complete block of the ilio-femoral region and the distal vessels are not often sufficiently open to allow an adequate flow of blood through the graft.

3. Amputation - The indications for amputation are similar in many respects to those outlined for the atherosclerotic group (see p. 420), although the approach tends to be somewhat more conservative from the point of view of the preservation of tissue. Most patients with Buerger's disease who are managed carefully do not require amputation. The results of amputation of the middle 3 toes are better than the results which can be achieved by amputation of the great and little toes. It is almost never necessary to amputate

the entire hand, although fingers must occasionally be removed.

If there is evidence of both large and small vessel disease, the results of conservative management are poor and amputation is frequently necessary. Pain may become so severe that the conservative approach must be discarded.

Prognosis.

Except in the case of the rapidly progressive form of the disease - and provided the patient stops smoking and takes good care of his feet - the prognosis for survival of the extremities is good. Thromboangiitis obliterans rarely results in death.

ARTERIAL EMBOLISM

Arterial embolism usually occurs as a complication of rheumatic heart disease, myocardial infarction, bacterial endocarditis, or congestive heart failure; about two-thirds of cases are associated with atrial fibrillation. In about 10% of patients no source of the embolus is clinically evident, and in this group the differential diagnosis between embolism and thrombosis may be difficult.

Emboli tend to lodge at the bifurcation of an artery and usually occur in the arteries of the lower extremities, although the arteries of the upper extremities, brain, or viscera are occasionally involved.

Clinical Findings.

In the extremity the initial symptoms are usually pain (sudden or gradual in onset), numbness, coldness, and tingling. Signs include absence of pulsations in the arteries distal to the block, coldness, pallor or mottling, hypesthesia or anesthesia, and weakness or paresis of the limb.

Treatment.

Immediate embolectomy is the treatment of choice in most cases. It should be done within 12 hours if possible.

A. Emergency Preoperative Care:
  1. Heparin sodium, 50 mg. ($3/4$ gr.) I.V., should be given as soon as the diagnosis is made or suspected in an effort to prevent distal thrombosis. The effect of this dose will usually be dissipated by the time the patient has been transported to the hospital and to surgery. If a 4-5 hour delay is anticipated, 30-40 mg. ($1/2$-$2/3$ gr.) should also be given I.M.
  2. A sympathetic block should not be attempted once heparin has been given, and it is better to emphasize heparin and surgery at the earliest moment rather than to risk the delay involved in a blocking procedure. If surgery must be delayed or if the patient is considered inoperable, a sympathetic block may be tried before heparin is given.
  3. Keep the extremity at or below the horizontal plane. Do not apply heat or cold to the involved extremity (but heat to an uninvolved arm may help produce a reflex vasodilatation). Protect from hard surfaces and overlying bedclothes.
B. Surgical Treatment:
  1. Use local anesthesia for embolectomy of a vessel of an arm

or leg; general anesthesia if the abdomen must be opened.

2. Extraction of the embolus - Control of the artery above and below the embolus must be achieved before extraction of the clot. Care must be taken not to dislodge the embolus to a more distal segment during the exposure of the artery.

Local arterial spasm, if present, is best treated by the application of a solution of papaverine hydrochloride, 2%.

A distal thrombosis may be removed by exposure of a more peripheral artery and retrograde irrigation with dilute heparin-saline solution at body temperature in an effort to wash the thrombus out through the proximal arteriotomy.

C. Postoperative Care: Because there is a marked tendency toward hematoma formation postoperatively, anticoagulants should be withheld for a few days.

Disappearance of a distal pulse in the early postoperative period usually requires reexploration in an attempt to remove the thrombus.

Prognosis.

Arterial embolism is a threat not only to the limb but also to the life of the patient. The operative mortality rate is about 25%.

Mortality increases with the size and location of the embolus; aortic and iliac emboli are the most dangerous. Concomitant cerebral or mesenteric embolism may occur, as well as progressive cardiac failure. Emboli associated with hypertensive or arteriosclerotic heart disease have a poorer prognosis than those arising from rheumatic valvular disease in younger patients. Emboli recur in almost half of the entire group of patients, and in over half of those with atrial fibrillation.

In patients with atrial fibrillation an attempt should be made to restore normal rhythm with quinidine, although this is usually possible only in patients with recent or transitory fibrillation. Long-term anticoagulant therapy may diminish the danger of further emboli, but in itself may lead to hemorrhagic complications. Correction of mitral stenosis and amputation of the atrial appendage in patients who have experienced previous emboli is associated with an appreciable incidence of operative or postoperative emboli, but this incidence is apparently lower than that which may be expected in patients with mitral stenosis and refractory atrial fibrillation who do not go to surgery. Surgery may therefore be justified in selected cases. Mitral valvuloplasties done in the earlier stages of mitral stenosis will diminish the chances of embolic complications.

VASOSPASTIC DISORDERS

RAYNAUD'S PHENOMENON AND DISEASE

Raynaud's phenomenon (secondary) or disease (primary) is characterized by intermittent attacks of pallor or cyanosis - or pallor followed by cyanosis - in the fingers (and rarely the toes) precipitated by cold or occasionally by emotion. Early in the course

of the disease, only 1 or 2 fingertips may be affected; as the disease progresses, all of the fingers down to the distal palm may be involved. The thumbs are rarely affected. General as well as local body cooling is usually necessary. Recovery usually begins near the base of the fingers as a bright-red return of color to the cyanotic or pale digit. Sensory changes which often accompany the vasomotor manifestations include numbness, stiffness, diminished sensation, and aching pain. If there is organic narrowing or obstruction of the digital vessels, the condition may progress to atrophy of the terminal fat pads and the digital skin, and gangrenous ulcers may appear near the fingertips which may heal during warm weather.

Raynaud's phenomenon may be primary, with no organic occlusion of the arteries, or may occur as a manifestation of severe or chronic cold injuries, vibration injuries, scleroderma and certain other collagen diseases, neurologic disorders affecting the arms, obliterative vascular disease of the atherosclerotic or thromboangiitis obliterans type, cervical rib syndrome or scalenus anticus syndrome, ergot poisoning, and other disorders.

Raynaud's disease is much rarer than Raynaud's phenomenon. It appears first between the ages of 25 and 45, almost always in women. Its earlier manifestations are similar to those described for Raynaud's phenomenon, but the disorder is progressive, with early trophic changes (skin atrophy or sclerodermatous changes, irregularity of nail growth, wasting of the finger pads, and small, painful, recurrent necrotic ulcers at the tips of the fingers which may in time lead to shortening of the fingers). Symmetric involvement of the fingers of both hands is the rule, and the spasm gradually becomes more frequent and prolonged. Gangrene of the whole finger is rare.

Treatment.
    A. General Measures: Avoidance of cold and injuries to the fingers is sufficient therapy for mild cases. Tolazoline (Priscoline®), 25-50 mg. ($3/8-3/4$ gr.) 3-4 times a day (or the longer acting tablet every 12 hours), may be of value.
    B. Surgical Treatment: Although the benefits of dorsal sympathectomy to dilate the cutaneous vessels of the digits are limited, this is still the most effective method of treatment for Raynaud's phenomenon or disease. Symptoms tend to recur in 2-5 years with the gradual return of sympathetic activity. Sympathectomy is of no value in advanced severe cases.

<h3 style="text-align:center">VASOMOTOR DISORDERS ASSOCIATED
WITH TRAUMA
(Sudeck's Atrophy, Causalgia)</h3>

Sudeck's atrophy is an acute atrophy of the bones of an extremity which usually comes on after minor injury, especially to the ankle or wrist. Symptoms and signs of vasomotor hyperactivity include pain of a burning type made worse by movement, edema, local heat, and swelling. The limb may ultimately become cold, cyanotic, and wasted, with stiffness of the joints. Secondary frac-

tures occasionally occur in the atrophic bones.

Prophylaxis consists of adequate early treatment of sprains. The early manifestations are usually treated by physical therapy: mild heat, light massage, and gentle movement of the joints. A walking type of plaster cast for the foot and ankle region may be of value.

In severe and chronic forms, sympathectomy may give relief.

Causalgia, which is characterized by intense burning pain and vasodilatation in an extremity, is a rare disorder caused by partial division or bruising of a peripheral nerve (usually the median nerve) or involvement of the nerve in scar tissue. The injury may be trivial. The pain is distal to the point of injury but not confined to the course of the nerve, and may not appear for a few days or weeks. Pain is initiated by light touch, temperature changes, or movement of the limb. The skin becomes red, smooth, devoid of wrinkles and hair, scaly, and cold, with disuse atrophy of the bones. If severe nervous and mental manifestations are prominent, local or operative procedures will probably be of no value.

Treatment.
  A. Conservative Treatment: Keeping the affected area cool and protected from stimuli is the treatment of choice even though the patient often demands more aggressive therapy, since the disorder usually subsides after a year or so.
  B. Surgical Treatment: Sympathectomy may be of value if a sympathetic block gives relief, and this is usually the treatment of choice if operative measures are required. Division of the nerve distal to the site of irritation gives relief but denervates the tissues. Reamputation of a painful stump is often followed by recurrence of symptoms in the new stump. Spinothalamic tractotomy is a desperate measure which is not always successful.

DEGENERATIVE AND INFLAMMATORY VENOUS DISEASE

VARICOSE VEINS

Varicose veins develop predominantly in the lower extremities, and consist of abnormally dilated, elongated, and tortuous alterations in the saphenous veins and their tributaries. These vessels lie immediately beneath the skin and superficial to the deep fascia and so do not have as adequate support as the veins deep in the leg, which are surrounded by muscles. In many cases there is an inherited defect in the walls or valves of the veins which leads ultimately to the formation of varicosities. Other contributory factors are prolonged standing over a number of years, pregnancy, obesity, and perhaps aging.

Secondary varicosities can develop as a result of obstructive changes in the deep venous system following thrombophlebitis, or occasionally as a result of proximal venous occlusion due to neoplasm. Congenital or acquired arteriovenous fistulas are also as-

sociated with varicosities.

The great saphenous vein and its tributaries are most common-
ly involved, but the small saphenous vein is occasionally affected.
There may be 1 or many incompetent perforating veins in the thigh
and lower leg, so that blood can reflux into the varicosities not
only from above, by way of the saphenofemoral junction, but also
from the deep system of veins through the incompetent perforators.
Largely because of these valvular defects, venous pressure in the
superficial veins does not fall appreciably on walking; over the
years the veins progressively enlarge and the surrounding tissue
and skin develop secondary changes such as fibrosis, chronic ede-
ma, and pigmentation. Atrophic changes take place in the skin.

Clinical Findings.

A. Symptoms: Extensive varicose veins may produce no subjec-
   tive symptoms, whereas minimal varicosities may produce
   many symptoms. Aching or burning discomfort, fatigue, or
   pain in the lower leg brought on by periods of standing are the
   most common complaints. Cramps may occur, but intermit-
   tent claudication and coldness of the feet are not associated with
   varicose veins. One must be careful to distinguish between the
   symptoms of arteriosclerotic peripheral vascular disease and
   those of venous disease, since occlusive arterial disease usu-
   ally contraindicates the operative treatment of varicosities.
   Itching from an associated eczematoid dermatitis may occur in
   the region of the veins.

B. Signs: Inspection is the best means of determining which veins
   are involved. Secondary tissue changes may be absent even in
   extensive varicosities; but if the varicosities are of long dura-
   tion, brownish pigmentation and thinning of the skin above the
   ankle are often present. Swelling may occur, but extensive
   swelling and fibrosis in the subcutaneous tissues of the lower
   leg usually denote the postphlebitic state.

C. Special Examinations:
   1. Percussion test with the patient standing - Percussion over
      the varicosities with 1 hand while palpating along the course
      of the vein with the other will show the course of the vari-
      cosities.
   2. Trendelenburg's test - Of use in determining the competence
      of the valves at the saphenofemoral junction and in the com-
      munications between the superficial and deep vessels.
      a. With the patient supine, elevate the leg. If there is no
         organic venous obstruction, varicosities will empty im-
         mediately.
      b. Place a rubber tourniquet around the upper thigh and ask
         the patient to stand up.
         (1) If the long saphenous vein remains empty or fills very
             slowly from below, the saphenofemoral valve is in-
             competent, the valves in the communicating veins are
             competent, and the blood is flowing through them in
             the right direction (superficial to deep).
         (2) If the varicosities fill rapidly, the communicating
             veins between the deep and the superficial vessels are
             incompetent and blood is refluxing into the varicosed
             vessels. The precise site of these defective perforating

veins can be determined by repetition of this maneuver, placing the tourniquet at successively lower levels. The sites of the incompetent perforators can be marked with a solution of pyrogallic acid and ferric chloride.

    c. Release the tourniquet. If the empty veins fill rapidly from above downward, incompetence of the saphenofemoral valve is confirmed.

  3. Perthes' test may be used to determine if the saphenous valves are competent, if the communicating valves are competent, or if deep venous obstruction is present. With the patient standing, a tourniquet is applied to the thigh so as to occlude the superficial but not the deep veins of the leg; the patient is then required to walk for 5 minutes.

    a. If the veins collapse, the communicating veins are competent. If, on releasing the tourniquet, the veins fill relatively slowly (35-60 seconds), the saphenofemoral valve is also competent.

    b. If the veins remain unchanged, the valves of the communicating and saphenous veins are incompetent and the pressure in the 2 systems is the same.

    c. If the veins become more prominent and pain develops, the deep veins are obstructed and the valves of the communicating veins may be incompetent.

Complications.

A. Ulceration will often result from a blow to the thin, atrophic, pigmented skin of the lower leg or ankle region, and such a lesion often becomes chronic, scarred, and severely painful. Treatment is with rest and elevation of the leg, and saline compresses. A skin graft may be needed. If the patient must remain ambulatory, firm compression of the lower leg and foot as afforded by Unna's boot or a compression boot dressing may be used. Recurrences are common.

B. Thrombophlebitis, starting in the varicosities and occasionally extending into the deep system, sometimes occurs.

Differential Diagnosis.

Primary varicosities should be differentiated from those secondary to obstruction or incompetence in the deep system of veins since surgery for the latter type of case is often of little or no benefit.

Treatment.

Varicosities tend to progress, and some form of therapy must be instituted if progressive changes and complications are to be avoided. Only surgery can provide prolonged relief from varicose veins when the saphenofemoral valve is incompetent.

A. Conservative Treatment:

  1. Elastic stockings and intermittent elevation of the legs is the best therapeutic approach in very old or poor risk patients, in patients who refuse surgery or have to put it off, and sometimes in women with mild or moderate varicosities who are going to have more children (since better long-term results can be obtained in women who are not going to have further pregnancies). Elastic stockings may also be of value

in those patients who already show a tendency toward vari-
cosities or whose families have a high incidence of varicosi-
ties, especially if they must spend many hours standing.

2. Injection treatment of varicosities with sclerosing solutions
to produce thrombosis of the segment of vein should be re-
served for treatment of short segments which remain after
surgery. The recurrence rate after injection therapy is
high, and complications can occur (e.g., local reactions
and infections around the vein, or deep thrombophlebitis).

Technic: A very fine (No. 25) needle is inserted into the
varix with the leg dependent; after the needle is inserted the
leg can be placed in the horizontal position. After drawing
blood back into the syringe to make sure the needle is in the
vein, and after the vein is relatively free of blood, 1-2 ml.
of sodium psylliate (Sylnasol®) or a similar sclerosing solu-
tion is injected. The needle is withdrawn, and digital pres-
sure is applied for 2-3 minutes above and below the area of
injection to hold most of the solution where its effect is most
desired. Only 2-3 areas should be injected at each office
visit

B. Surgical Treatment: High ligation at the saphenofemoral junc-
tion, with stripping of the vein and interruption of incompetent
perforators, is the treatment of choice. Multiple low ligations,
taking care to interrupt all incompetent perforating veins as
well as the major channels, will also give good results if done
thoroughly. Postoperative discomfort is usually less with this
procedure, and it can be done under local anesthesia. For
these reasons it is usually the method of choice in older or
poor risk patients or when there is an open, chronically infec-
ted ulcer.

C. Postoperative Care: Ambulation with elastic bandages is en-
couraged on the day of surgery if local anesthesia is used, or
the next day if general anesthesia is used. Standing or sitting
is not desirable, and when the patient is in bed the leg should
be elevated.

Prognosis.

All patients should be informed that even thorough and extensive
surgery may not remove all varicosities and that additional (more
limited) procedures may be necessary either in the early postop-
erative period or months or years later. If extensive varicosities
reappear after surgery, the completeness of the high ligation should
be questioned and reexploration of the saphenofemoral area may be
necessary.

DEEP THROMBOPHLEBITIS

Thrombophlebitis is a partial or complete occlusion of a vein
by a thrombus with a secondary inflammatory reaction in the wall
of the vein. It is encountered most frequently in the deep veins in
the legs and pelvis in postoperative or postpartum patients between
the fourth and the fourteenth days, and in patients with fractures
and cardiac disease.

The deep veins of the calf are most frequently involved, but the

thrombotic process may progress to involve the femoral and iliac veins. The site of origin is at times in the pelvis or in the great saphenous vein.

Predisposing factors are aging, malignancy, anemia, and chronic infection. Perhaps the most prominent etiologic factors in thrombophlebitis are venous stasis and the pressure changes produced in the endothelium of the vein wall as the legs lie for hours supported by the mattress of the bed or operating table. Impairment of the coagulation mechanism may play a role, and other poorly understood factors are probably also involved.

Clinical Findings.

There may be no symptoms or signs in the extremity in the early stages. The patient not infrequently suffers a pulmonary embolus, presumably from the leg veins, without demonstrable abnormalities in the extremities.

A. Symptoms: There may be no symptoms, or the patient may complain of a dull ache, a tight feeling, or frank pain in the calf or, in more extensive cases, the whole leg.

B. Signs: Typical findings, though variable, are as follows: tenderness and induration or spasm in the calf muscles; slight swelling in the involved calf, as noted by careful measurements; pain in the calf produced by dorsiflexion of the foot (Homan's sign); and slight fever and tachycardia. When the femoral and iliac veins are also involved, the swelling in the leg may be quite marked. The skin may be slightly cyanotic if venous stasis is severe, or pale and cool if a reflex arterial spasm is superimposed.

Complications.

A small or moderately large pulmonary embolism may be present without any associated pulmonary symptoms, signs, or x-ray findings. Clinical manifestations, when present, take the form of pleuritic pain, often with a transient friction rub; a dry cough, sometimes associated with hemoptysis; local rales in the area of involvement; a small amount of pleural fluid; and, occasionally, x-ray evidence of pulmonary consolidation. Fever and increased pulse and respiration rates are frequently present.

Massive pulmonary embolism is associated with shock, dyspnea, and cyanosis, and often with transient Ecg. changes characteristic of cor pulmonale. Death may occur in minutes or hours.

Treatment.

A. Local Measures and Follow-up Care:

1. Elevation of the legs by means of absolute bed rest with the foot of the bed on six-inch blocks and the head of the bed in the horizontal position is maintained during the initial period. After 7-10 days, when the inflammatory aspects have had time to produce a more adherent thrombus - and provided the swelling and local symptoms have largely subsided - walking but not standing or sitting is permitted.

2. Elastic bandages or stockings are applied from the toes to just below the knees as soon as the diagnosis is made and continued for at least 6-12 months. Bandages and anticoagulants initially, and intermittent elevation of the legs subsequently, will do much to reduce the danger of permanent

changes and disability. These measures are perhaps even more important if surgical ligation of the femoral vein or the vena cava has been necessary.

B. Medical Treatment (Anticoagulants): (See p. 608.) Anticoagulant therapy is considered to be definitive in most cases of deep thrombophlebitis with or without pulmonary embolism. There is good evidence that the relatively high incidence of fatal pulmonary embolism secondary to venous thrombosis is significantly reduced by adequate anticoagulant therapy. Progressive thrombosis with its associated morbidity is also reduced considerably, and the chronic secondary changes in the involved leg are probably also less severe. The specific drug used depends to some extent on the physician's preferance and experience, but the relative urgency of the situation usually requires the use of heparin or 1 of the short-acting coumarin compounds at least in the initial phase of treatment; after a few days 1 of the longer acting drugs can be used.

Anticoagulants should be continued for at least 3 days after subsidence of local pain and tenderness in the involved extremity and until ambulation has been fully established. The rate of subsidence of symptoms is variable, and occasional cases are quite refractory to therapy. Therapy should be continued for at least 10 days for venous thrombosis and 21 days for pulmonary embolism, but therapy may have to be continued for a longer period if signs and symptoms persist.

C. Surgical Treatment: Surgical division of both common femoral veins or, if the iliac and pelvic veins are also involved, the inferior vena cava must be strongly considered if a patient on adequate anticoagulant therapy has a pulmonary embolism. Although some degree of chronic edema of the legs may develop as a result of ligation, it can usually be minimized if anticoagulant therapy is resumed following surgery and if follow-up care, consisting of elastic supports to the lower legs and elevation of the legs at intervals during the day and at night, is continued for at least 1 year. The chances of a second, possibly fatal pulmonary embolism are appreciably reduced after ligation of both femoral veins, and ligation of the inferior vena cava is almost sure to prevent further emboli.

Prophylaxis.

A. Pressure on the calf or thigh during a long operation should be avoided.

B. Patients with a history of phlebitis or with varicose veins should have elastic supports on the legs during and after operation.

C. Preoperative correction of anemia, dehydration, congestive failure, or metabolic disturbances will diminish the incidence of thrombophlebitis.

D. Postoperative exercises of the legs should be started at the close of the operative procedure while the patient is still on the operating table and continued for several days after operation. Early ambulation (but not standing or sitting) is likewise of value.

E. Elevation of the foot of the bed with the head of the bed in a horizontal position may be of value in patients predisposed to thrombophlebitis. Deep breathing and frequent turning should be encouraged.

SUPERFICIAL THROMBOPHLEBITIS

Superficial thrombophlebitis may occur spontaneously, as in pregnant or postpartum women or in individuals with varicose veins or thromboangiitis obliterans; or it may be associated with trauma, as in the case of a blow to the leg or following intravenous therapy with irritating solutions. It may also be a manifestation of abdominal malignancy such as carcinoma of the pancreas. The great saphenous vein is most often involved. Superficial thrombophlebitis is usually not associated with thrombosis in the deep leg veins. Pulmonary emboli are infrequent but do occur.

Clinical Findings.

The patient usually experiences a dull pain in the region of the involved vein. Local findings consist of induration, redness, and tenderness over the vein. The process may be localized, or it may involve most of the great saphenous vein and its tributaries. If the process is migratory, involving 1 segment after another, thromboangiitis obliterans should be suspected.

Treatment.

If the process is well localized and away from the saphenofemoral junction, treatment consists of moist heat to the area and bed rest in the "legs elevated" position. Anticoagulants are usually not indicated except when the process seems to be very active and there is fear of involvement of the deep system.

If an appreciable segment of the greater saphenous vein is thrombosed, or if the process shows a tendency to progress up the leg and thigh, ligation and division of the saphenous vein at the saphenofemoral junction is indicated. The manifestations usually regress following this procedure.

CHRONIC VENOUS INSUFFICIENCY

Chronic venous insufficiency commonly results from changes secondary to deep thrombophlebitis (the postphlebitis syndrome), but it can also occur as a result of neoplastic obstruction of the pelvic veins or congenital or acquired arteriovenous fistulas.

When insufficiency is secondary to deep thrombophlebitis, the valves in the deep venous channels and sometimes in the perforating veins as well have been destroyed by the thrombotic process and the vessels are often irregular and tortuous. The venous pressure therefore fails to drop when the patient walks; and because the standing venous pressures are not reduced, secondary changes eventually take place in the venules, capillaries, subcutaneous tissues, skin, and superficial veins.

Clinical Findings.

Chronic venous insufficiency is characterized by progressive edema of the leg (particularly the lower leg) and secondary changes in the skin and subcutaneous tissues. The usual symptoms are itching, a dull discomfort made worse by periods of standing, and pain if an ulceration is present. The skin is usually thin, shiny, atrophic, and often brownish. Eczema is often present, and there

may be superficial weeping dermatitis of large areas. The subcutaneous tissues are thick and fibrous. Recurrent ulcerations are common, usually just above the ankle; healing results in a thin scar on a fibrotic base. Varicosities often appear and are usually associated with incompetent perforating veins in the mid and upper part of the lower leg.

Treatment.
 A. Bed rest with the legs elevated to diminish chronic edema is fundamental to the treatment of chronic venous insufficiency. Subsequent measures to control the tendency toward edema include (1) intermittent elevation of the legs during the day and elevation of the foot of the bed at night; (2) avoidance of long periods of sitting or standing; and (3) the use (for life) of well-fitting elastic supports worn from the toes to just below the knee.
 B. Chronic eczema should be treated by strict avoidance of scratching and a bland, nonirritating application such as zinc oxide ointment, calamine lotion, or moist applications of 1:10,000 potassium permanganate solution.
 C. Subacute or chronic cellulitis may respond very slowly to rest and local heat.
 D. Ulcerations are preferably treated with compresses of isotonic saline solution, which aid in the healing of the ulcer or may help prepare the base for a skin graft; or the lesion can be treated on an ambulatory basis by means of a boot of Unna's paste on the lower leg after much of the swelling has been reduced by a period of elevation.
 E. Varicosities secondary to the obstructive elements and the valvular defects in the deep system of veins may in turn contribute to the undesirable changes in the tissues of the lower leg. Varicosities should occasionally be removed, but the tendency toward edema will persist and the measures outlined above will be required for life. In general, the varicosities can be treated along with the edema by nonoperative means. If the obstructive element in the deep system is severe, it may be most undesirable to remove superficial channels which may be carrying most of the blood out of the leg. (See Perthes' test, p. 432.)

Prophylaxis.
 Irreversible tissue changes and associated complications in the lower legs can be prevented through adequate treatment of acute thrombophlebitis with anticoagulants and energetic measures to avoid edema in the subsequent years by means of elastic supports to the lower legs, intermittent periods of elevation of the legs, and elevation of the foot of the bed.

LYMPHEDEMA

The underlying mechanism in lymphedema is impairment of the flow of lymph from an extremity, either as a result of an inflammatory or noninflammatory obstruction of the channels or because of

malformations in the vessels themselves. Secondary dilatation leads to incompetence of the valve system, disrupting the orderly flow along the lymph vessels and resulting in progressive stasis of a protein-rich fluid with secondary fibrosis. Episodes of acute and chronic inflammation may be superimposed, with further stasis and fibrosis. Hypertrophy of the limb results, with markedly thickened and fibrotic skin and subcutaneous tissue and diminution in the fatty tissue.

Treatment.

There is no very satisfactory treatment for lymphedema, but the following measures can be instituted: (1) The flow of lymph out of the extremity, with a consequent decrease in the degree of stasis, can be aided through intermittent elevation of the extremity, especially during the sleeping hours; and with elastic bandages or supports and massage toward the trunk. (2) Secondary cellulitis in the extremity should be avoided by means of good hygiene and treatment of any trichophytosis of the toes. Once an infection starts, it should be treated thoroughly. (3) Surgical excision of strips of skin and edematous subcutaneous tissue down to the fascia in staged procedures - or even an extensive excision and grafting procedure - can be considered in the very chronic and severe forms, but the cosmetic and functional results leave much to be desired and elastic supports will still be necessary for life.

16 . . .

Plastic Surgery

The objective of plastic and reconstructive surgery is the repair of congenital or acquired defects to improve function and appearance. The plastic surgeon is called upon to treat a wide variety of deformities, limited for the most part to the surface tissues but including also the deeper structures of the head, neck, and hands.

Planning a Reconstructive Operation.
    A. Make an accurate diagnosis of the deformity. From the standpoint of plastic surgery, this includes a determination of the amount of tissue loss and the degree of distortion, separation, and atrophy or hypertrophy.
    B. Consider all possible methods of repair and select one which will achieve the best possible result in the simplest and most direct manner, utilizing neighboring tissues whenever possible.
    C. In multiple stage procedures, plan in detail the entire sequence in advance. Pedicle grafts which are to be transferred should be designed to avoid torsion or unnecessary discomfort during all stages of healing.

Principles of Technic.
    Gentleness in handling tissues is a very important factor in successful reconstructive surgery; the selection of instruments, suture materials, and procedures is based on this principle.
    A. Incisions are made at right angles to the surface, not on the bevel. They should not cross normal flexion or expression creases, but should conform to Langer's lines (see p. 441) whenever possible.
    B. Hemostasis must be meticulous. Do not grasp more tissue than necessary with fine mosquito forceps. Use fine ties of 000 or 0000 catgut or the electrocautery.
    C. Do not manipulate the skin with forceps; grasp the subcutaneous tissue with fine-tooth forceps or skin hooks.
    D. Subcutaneous tissues are closed with fine plain catgut and knots are tied on the deep surface. Avoid tension in closing. Undermine in the subcutaneous plane only as necessary to permit relaxed closure. (See p. 440.)
    E. Skin sutures are placed for accurate approximation after the wound edges have been brought together with catgut. The simplest suture is preferred: interrupted, vertical or horizontal mattress, or subcuticular, according to the situation and the surgeon's preference. Use 00000 or 000000 silk or nylon with an atraumatic needle. The needle should enter and emerge at right angles, close to the wound margins. Sutures should not be tied tightly, to avoid cross hatching.

Sources of Grafts.
    A. Autogenous: Taken from the patient. May be skin, bone, cartilage, fat, or fascia.
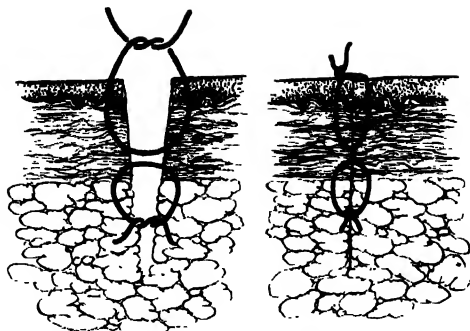
Wound Closure in Layers

Continuous Horizontal Mattress Sutures
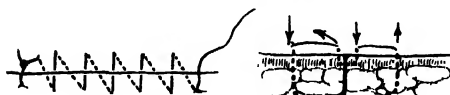
Continuous Vertical Mattress Sutures
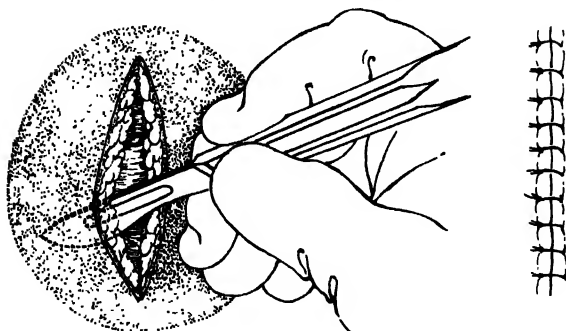
Undermining and Wound Closure. Stippling shows area to be undermined. At right, closure of incisions by interrupted sutures.
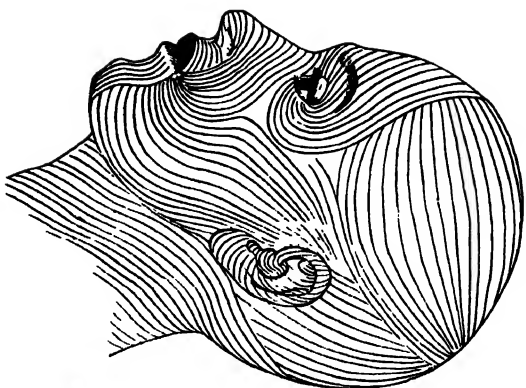
Side of Face
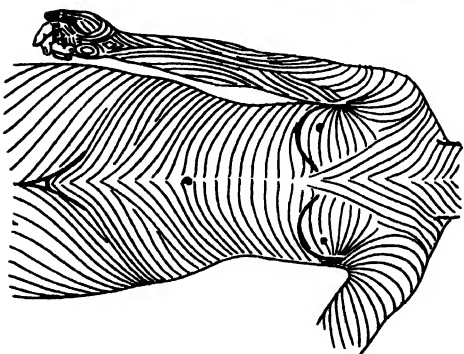
Anterior View of Trunk
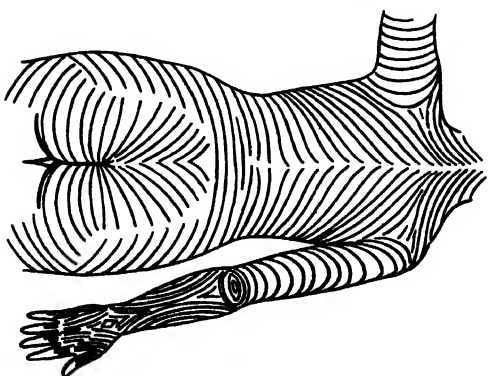
Posterior View of Trunk

Langer's Lines, Showing the Lines of Skin Tension

Langer's Lines 441

B. Heterogenous: Taken from a source other than the patient.
  1. Isografts (homografts) - Taken from the same species.
     (Bone, cartilage, cornea.) Skin isografts are used as a
     temporary covering in extensive burns but will not survive
     unless taken from an identical twin.
  2. Zoografts - Taken from other animal species. Not used
     at the present time.

SKIN AND PEDICLE GRAFTS

Types of Free and Pedicle Skin Grafts.
A. Free Skin Grafts: These are grafts which are completely re-
   moved from 1 part of the body and applied to another part.
   They are used to replace skin losses only, and their survival
   requires a recipient site with a healthy vascular bed. They
   must therefore not be applied to areas devoid of adequate cir-
   culation, on exposed bone or tendon, or on grossly infected
   wounds. Free grafts are occasionally used to replace large
   mucous membrane losses, e.g., as a lining for a reconstructed
   nose. Free skin grafts are of 2 kinds:
  1. Split-thickness (Thiersch) - Split-thickness grafts may be
     taken from the extremities, abdomen, or back for covering
     granulating defects, facial defects (temporary or permanent
     covering), for resurfacing the hands, or for inlays. If
     split-thickness grafts are cut too thin they will contract and
     wrinkle, but thicker grafts make a good surface covering.
     Color match is poor. The donor site heals spontaneously.
  2. Full-thickness (Wolfe) - Full-thickness grafts may be taken
     from the postauricular area, the neck, or the supraclavicu-
     lar region for repair of facial defects or replacement of
     eyelid tissues. They may be taken from the extremities,
     the iliac crest, or the abdomen for replacement of the volar
     surface of the hand. Secondary contraction does not occur,
     and color match is usually good. The donor site requires
     grafting.
B. Pedicle Grafts or Flaps: These contain skin and subcutaneous
   tissue, and must retain a circulatory connection through an
   attachment at 1 or both ends. They are used when free grafts
   are inadequate; to cover exposed bone or tendon; and in instances
   demanding not only skin but subcutaneous tissue replacement as
   well. The pedicle attachment must be maintained until a new
   circulation has developed from the recipient site; this usually
   requires about 3 weeks.
   ' A pedicle graft may be raised and transferred at 1 opera-
   tion, or, if there is any doubt concerning the arterial or venous
   circulation, a "delaying" procedure may be done. To "delay"
   a flap means to incise and partially raise the tissue, then re-
   place and suture it in its original site. The objective is not
   only to test the circulation but to encourage increased blood
   supply from the pedicle, which will eventually be its only con-
   nection.
   The circulation of a flap depends upon its arterial supply and
   its venous return. Should the former be deficient, the flap will
   become white; if the latter is inadequate, the flap will become
   blue and edematous, eventually strangulating its circulation

entirely. With this in mind flaps should be designed so that the best possible circulation is afforded. The base should be broad, and the length should ordinarily not exceed two and one-half times the width.

Pedicle grafts or flaps are of 2 kinds:

1. Direct pedicle flaps - These are designed, raised, and shifted as a unit. The flap may utilize local neighboring tissues and be shifted or rotated into its new location; or it may be transferred from a distance, as in the case of an abdominal flap transported to the neck by way of an intermediate attachment to the wrist.

2. Tubed pedicle grafts - These form a closed unit which is migrated by shifting first 1 end of the pedicle and then the other until the graft has reached its destination. An interval of at least 3 weeks is usually necessary between stages. This type of graft requires an additional stage for the construction of the tube. However, because it is a closed unit with no raw surface, the chances of infection and subsequent shrinkage of the graft are minimized and dressings and hospitalization are reduced. The tubed pedicle graft is most often used when multiple transfers are required.

Preparation of a Granulating Wound for Skin Grafting.

A. Debridement: Much time may be saved by mechanical debridement under general anesthesia rather than waiting for gradual separation of the eschar. This seems more satisfactory and is more rapid than enzymatic debridement, which depends upon gradual digestion of necrotic tissue.

B. Interim Management:

1. Wet dressings of saline will aid in the preparation of the wound. These should be changed at least every 12 hours for best results. Acetic acid, 0.5-1%, may be added to help control the growth of pseudomonas organisms.

2. Antibiotics locally or systemically may be indicated after determination of sensitivity. These drugs should not be continued over prolonged periods.

Procedure at Time of Grafting.

A. General anesthesia is most often used. Local anesthesia may be used if the area to be grafted is small. In this case the donor area is infiltrated with 0.5-1% lidocaine (Xylocaine®) containing epinephrine, 2-4 drops/oz. The recipient area should not be infiltrated, since the wound is not sterile and infiltration may contaminate the surrounding tissues.

B. A previously selected donor site is then exposed. This site should be chosen with the following in mind:

1. If the Thiersch knife is to be used a flat surface is required, preferably the hips or thighs or the medial aspect of the arm. A flat surface is also required for the Brown electrodermatome, but in this case the graft may also be taken from the back. If the Padgett or Reese dermatome is used, the abdomen is also a suitable donor site. Saline may be injected into the tissues of the donor site if bony prominences present a problem.

2. Hair is not a problem since in taking split skin grafts the follicles remain in the donor area.

3. In young females, avoid using the thighs or other areas which would normally be exposed in play clothes (unless no other area is available). Some scarring, discoloration, or depigmentation is likely to remain after healing.

C. Prepare the donor area by thorough rubbing with hexachlorophene for 5 minutes followed by application of Zephiran® or a similar antiseptic. Drape and cover the donor area. Prepare well around the recipient site in a similar way, using only saline irrigations for the wound itself. Drape and cover the recipient area.

D. Estimate as accurately as possible the total amount of skin required, and cut the grafts to avoid the hazard of returning to the donor area after working at the recipient site. The depth of the grafts will vary from 0.008 to 0.014 inches in children and from 0.014 to 0.024 inches in adults. Spread the grafts on gauze moistened with saline and cover with gauze. Place the grafts on a table where they will not be disturbed.

E. Dress the donor area and do not return to it.

F. If the granulations are healthy, firm, and flat, they need not be removed. If they are spongy or pale, or if there is considerable deep scarring, the granulations and underlying scar should be removed by scraping with a No. 10 Bard-Parker blade or by actual dissection. If the wound is large and involves an extremity, debridement may be done while bleeding is controlled with a tourniquet; bleeding is thereafter controlled by firm pressure for 5 minutes after the tourniquet is released. If a tourniquet is not used, the bleeding can be controlled by pressure, but blood replacement should be available.

G. Apply the grafts to the dry surface of the wound and spread out under normal skin tension. Suture the grafts to the skin edges with 0000 silk. It is not necessary to puncture the grafts for drainage. After suturing, make certain that no blood has accumulated under the graft. Apply vaseline or xeroform gauze, cover with several layers of flat smooth gauze and a layer of fluffed gauze, and wrap firmly with elastic bandage. Immobilize adjacent joints with a splint or cast. Elevate the part if it is an extremity, and do not disturb the grafts for 8-10 days unless there is drainage or prolonged pain.

An alternative method of management in selected cases (and particularly in chronic contaminated wounds) is to eliminate dressings entirely, protecting the area with a wire mesh and permitting direct visualization at all times. Any secretions around or under the grafts may then be gently expressed with applicators at once. In many instances grafts which would otherwise be lost may be saved in this way.

H. Any extra skin from the donor area should be rolled in vaseline gauze and placed in a test tube or glass jar in the refrigerator until the first dressing of the graft.

Dressing the Graft.

On the eighth to the tenth day, remove the splints and dressings and expose the grafts, taking care not to pull them away from the recipient area. Remove sutures. Viable skin will be pink; nonviable skin will be gray. Remove nonviable skin and gently cleanse

the area with saline. If a portion of the graft is not viable and the wound is clean, apply the refrigerated skin at the time of the first dressing or a few days thereafter. Reapply dressings and splint again for another week, changing dressings as required. After 2 weeks the grafts should be well enough established so that immobilization is no longer necessary.

Transplantation of Free Skin Grafts.

The free skin graft (as opposed to the pedicle graft, which carries its own blood supply) depends upon the recipient bed for its nourishment until a new blood supply has been established. This nourishment is provided by the tissue fluids, which serve to keep the graft viable until the upward growth of capillaries establishes circulation. The thinner the graft, the more rapidly is new circulation established. It is thus essential that the entire graft be in contact with the recipient bed. A hematoma or purulent drainage under any portion of the graft will interfere with the upward growth of capillaries, and that portion will therefore fail to survive. Meticulous hemostasis, avoidance of infection, and immobilization are prerequisites for successful grafting.

Healing and Innervation.

Healing of the graft is similar to wound healing elsewhere. After 2 weeks, the graft is firmly attached to the bed by connective tissue. A pliable layer of fat will usually be deposited in 3-4 weeks. In 4-6 weeks, sensory nerves will begin to grow in from the periphery; the sense of touch and the perception of pain and temperature appear in that order.

Treatment of Donor Site.

Avoid infection, which will delay healing and may even destroy remaining epithelium and necessitate grafting of the donor site. Two methods are available, either of which will achieve the desired result if contamination of the donor area is avoided.

A. Open Method: Apply a cradle over the wound to prevent contact with bed clothes and allow a natural crust to form. Crusting usually seals off the area in 24-48 hours and separates in 10-14 days.

B. Closed Method: Apply very fine mesh over the wound, such as silk or vaseline gauze. Apply a pressure dressing and allow it to remain in place for 10 days. If healing is delayed, apply 5% scarlet red ointment to stimulate the growth of epithelial cells. In case of contamination with drainage, apply wet saline dressings. Healing usually occurs in 14 days.

CONGENITAL ANOMALIES

CLEFT LIP AND PALATE

Approximately 1 out of every 750 infants is born with a cleft of the lip or palate (or both). Heredity is a definite etiologic factor. If either parent or a sibling is so affected, the unborn child has a

5% chance of also being affected; when both a parent and a sibling are affected, the risk rises to 15%. Maternal malnutrition, particularly vitamin and mineral deficiencies, and hormonal lack appear also to play a definite role in etiology.

Embryologically these clefts result from a failure of fusion of the various processes involved in the formation of the lip and palate, which ordinarily are complete by the tenth week of gestation.

Types of Clefts.

The cleft of the lip may vary from a small notching at the vermilion border to a complete separation involving the floor of the nose, with wide distortion of the nostril. The involvement may be unilateral or bilateral. In the case of the bilateral cleft, the premaxilla may be projected forward to a marked degree. Similarly, clefts of the palate may range from a split uvula or notching of the alveolus to a complete cleft into the nose. These clefts also may be unilateral or bilateral.

Treatment.

A. Timing of Operation: Closure of the cleft lip is usually done before the infant leaves the hospital; the age of 3 weeks is acceptable if the child is gaining normally.

Operation on the cleft palate is ordinarily done when the child is 12-18 months of age. Most surgeons believe the cleft should be closed before the child learns to speak; others prefer to delay the operation because of possible ill effects on maxillary growth centers.

B. Feeding: Prior to closure of the lip, it may be necessary to assist feedings if the infant has difficulty in sucking; this is most often noted in complete bilateral clefts. A Breck feeder may be used or, in extreme cases, gavage.

After the lip repair, gavage is usually employed for the first week; thereafter, regular bottle feedings are started.

C. General Principles of Repair:

1. Cleft lip - There is no actual tissue loss in cleft lip; the problem is to re-form the available lip tissue, which is present but separated. Numerous procedures have given excellent results in experienced hands, notably the procedures of Le Mesurier, Blair and Brown, and Tennison. Incisions are planned to give a full, normal appearing lip, correcting much of the nostril deformity at the same time.

2. Cleft palate - In the repair of palatal clefts it is not necessary to obtain bony union but merely to shift the palatal mucoperiosteum to the midline and suture it in place. The objective is not only to obtain closure of the cleft but to ensure adequate length of the soft palate for proper velopharyngeal closure. The procedures of Langenbeck, Wardill, and Veaux, or modifications of them, are most frequently used.

Prognosis.

With refinements in the technic of lip repair, it is possible to obtain excellent results in unilateral cases in 1 stage. Bilateral repairs are complicated by the protruding premaxilla and prolabium. A satisfactory result may be obtained in these cases, but it is usually necessary to resort to secondary procedures.

Cleft of Soft Palate Only

Complete Cleft of Soft Palate and Incomplete Cleft of Hard Palate

Complete Cleft of Palate

Bilateral Cleft Palate With Protruding Premaxilla

Complete Unilateral Cleft Lip

Complete Bilateral Cleft Lip

The results of cleft palate repair are evaluated on the basis of function. A normal appearing palate may not produce the best speech, and vice versa. Speech training should be begun at the age of 3 years, or as soon as the child will cooperate.

Patients with cleft lip or palate should be brought before a Cleft Palate Panel so that long-term care and follow-up can be planned. Such a panel should consist of the following: (1) dental consultants (dentists, orthodontists, prosthodontists, and pedodontists), (2) plastic surgeons, (3) pediatricians, (4) otologists and otolaryngologists, (5) audiologists, (6) speech therapists, and (7) psychologists, social workers, and vocational advisors.

Secondary and Supplementary Procedures.

    A. Nose and Lips: Revision of the nostril deformity may be required. The usual defect is a flattening of the nostril on the

cleft side. Minor revision of the vermilion border or cupid's bow may be desirable. In the case of bilateral clefts of the lip, a revision of the nose will probably be necessary since the columella nasi is shortened and the nasal tip is thus pulled downward. Occasionally, in addition to the nasal deformity, the upper lip will be tight and the maxilla underdeveloped. In such cases the rotation of the central portion of the full lower lip into the central portion of the upper lip will produce marked improvement (Abbé-Estlander operation).

B. Palate and Pharynx: Secondary procedures on the palate are indicated when the speech impediment is not correctible by speech therapy. Shortening of the palate with inadequate velopharyngeal closure is the primary cause of the typical cleft palate speech.

Dorrance's method of lengthening the palate consists of raising the entire palate and ligating the posterior palatine arteries at the first stage, and raising and pushing back the palate at the second stage. The VY method has also been used for many years, although the palate cannot be greatly lengthened in this way. In general, these operations do not produce sufficient improvement, since healing and scar contraction counteract some of the added length.

The operation of pharyngeal flap is more satisfactory in this regard. This consists of raising a flap on the posterior pharyngeal wall, usually based superiorly. The flap is then attached to a prepared donor site in the posterior border of the soft palate. On healing, the scar contraction draws the palate posteriorly, which is desirable.

If possible, secondary procedures should be concluded by school age to avoid psychic trauma.

PROTRUDING EARS

This defect is usually bilateral. The auricle may simply protrude from the side of the head to an extreme degree, may be cup-shaped in addition, or the superior border of the helix may overhang ("lopear"). The antihelix is usually not developed, and the auricular cartilage may be misshaped

Taping the ears in infancy is of no value. Surgery is the only treatment, and should be performed at about 5 years of age (preschool) to avoid psychologic trauma.

Treatment consists of correcting the abnormality of the cartilage through a postauricular incision. The antihelix is formed by parallel incisions 1 mm. apart along the normal line of the fold. The cartilage is then folded back upon itself and sutured, forming the rounded antihelix. Occasionally a separate incision is required along the anterior superior helix to remove or reshape the cartilage in this location.

MICROGNATHIA

Receding lower jaw may be combined with other congenital defects, especially cleft palate. Serious respiratory and feeding difficulties will occur in extreme cases as a result of posterior displacement of the tongue.

There should be no delay in treatment once the diagnosis has been made. Tracheostomy is sometimes indicated. The operation described by Douglas (Plast. & Reconstruct. Surg. 1:300, 1946) has been successful in many cases. This consists of suturing the tongue to the buccal surface of the lower lip. With subsequent mandibular growth, the tongue may usually be released in 3-4 months.

NEOPLASMS OF THE SKIN

CAPILLARY HEMANGIOMAS

Port Wine Stain (Nevus Flammeus).

These are usually flat, and vary in color from faint pink to bright red. Nodular outgrowths may occur. Port wine stains may involve any area of the body surface, but they are usually not a problem unless they occur on exposed areas.

These nevi do not regress. Small lesions may be surgically excised and the wound closed by direct approximation or local flaps. Larger lesions would necessitate full-thickness skin grafting, and it is usually preferable to leave them alone (relying on cosmetics to cover them). Tattooing has been used, but a good color match is difficult to obtain.

Port wine stain is not radiosensitive. Local applications of CO_2 snow may only lead to scarring.

Strawberry Birthmark (Hemangioma Simplex).

Strawberry marks are circumscribed, finely lobulated, and elevated, with dilated capillaries, veins, and arterioles (resembling a strawberry). The greater portion may be below the surface. They are most commonly seen on the face, neck, and scalp, but may occur anywhere on the body or face. They may ulcerate as a result of rapid growth or trauma.

Spontaneous regression occurs in a high percentage of cases during the first 7 years (not afterward). However, all do not regress, and even among those which do there may first be a period of rapid growth and ulceration with considerable deformity. A conservative attitude is usually to be recommended; however, in certain cases which show signs of active growth, many surgeons are of the opinion that surgical excision is indicated before the case is complicated by extensive increase in size and ulceration.

Strawberry birthmarks are radiosensitive, but the side effects of radiation must be carefully considered; excessive radiation may produce atrophy of the skin and underlying soft tissues, telangiectasis, or arrested development of bony structures.

If it is desirable to wait for regression to occur, injection of sclerosing substances such as sodium morrhuate or sodium psylliate

(Sylnasol®) may initiate regression.

Application of CO_2 snow has been used in the treatment of these lesions; however, the effect is inadequate since CO_2 snow blanches out the surface tissues only.

Cavernous Hemangiomas.

These are soft compressible tumors, predominantly venous in character, which contain thin-walled sinus spaces. They may be entirely subcutaneous, in which case the overlying skin usually shows a bluish discoloration, or they may involve skin as well. On the face they cause extensive deformity as well as disturbance of function. Complications (ulceration, infection, and hemorrhage) may be extremely serious.

The endothelium of the sinuses is sensitive to radiation, which initiates interstitial fibrosis and obliteration of the spaces. External radiation, however, may have undesirable side effects such as skin changes or disturbance of bone growth. Radon seeds have proved to be of great value in regulating the dosage to a given area. The usual dosage per seed is 0.1 millicurie. The seeds are inserted through small stab incisions at the periphery of the tumor, and should lie about 1-1.5 cm. apart in all planes.

Sclerosing solutions such as 5% sodium morrhuate may be injected into the tumor. This results in obliteration of the area by irritation and fibrosis. The solution need not be injected into a space or the lumen of a vessel since the obliterative reaction may be initiated anywhere in the tumor. Dosage varies from a few minims to 0.5 ml., depending upon the size of the growth. A marked tissue reaction will result from larger doses, and since more than 1 treatment will usually be required it is well to start with the smaller dose and evaluate the result. An interval of several weeks should elapse before repeating the injection.

Smaller lesions may be simply and effectively treated by surgical excision. Surgery is also frequently indicated following obliteration of these tumors by other methods to improve the cosmetic result.

Cirsoid Angiomas.

These are of the cavernous type, but they also have an arterial circulation by way of arteriovenous fistulas. They pulsate and present a bruit, and may bleed profusely.

Ligation of efferent vessels should precede any other treatment. This is done around the periphery, since ligation of main trunks will not be effective because of abundant collateral circulation. Following ligation, treatment is directed according to the methods describe for cavernous hemangiomas.

MELANOMAS

Melanomas are benign or malignant tumors composed of nevus cells which have the potentiality of forming melanin.

Diagnosis.
A. Pigmented Nevus:
    1. Intradermal (resting) - These are circumscribed, smooth or

slightly elevated, verrucous lesions. They vary in color from light to dark brown, and may contain hairs. The nevus cells are located in the dermis.

2. Junctional (active) - These are flat or slightly raised, smooth, and devoid of hairs. In color they vary from light brown or brownish black to slate blue or blue black. There is an active formation of nevus cells in the basal layer of the epidermis (dermo-epidermal junction). Many intradermal nevi show some areas of junctional activity. Junction nevi are potentially malignant.

B. Malignant Melanoma: These tumors arise invariably from junctional or compound nevi. At first there may be evidence of growth of the lesion or increase in depth of pigmentation. An inflammatory border may develop. In the later stages the lesion may fungate or ulcerate. Pigmentation is not essential to diagnosis (amelanotic melanoma). Satellites may appear in the neighboring skin. Metastasis usually occurs first through lymph channels, and later through the blood stream to the liver, lungs, brain, and skin.

These tumors are rare before puberty; when they do occur in this age group they are often clinically benign.

C. Blue Nevus: Blue nevi are sharply circumscribed, slate blue or bluish-black, round or oval lesions, usually measuring only a few mm. to 1 cm. in diameter. Malignancy does not seem to occur. The pigment is in the middle or lower third of the corium, and the epidermis is normal.

Treatment and Prognosis.

Pigmented moles which are subject to chronic irritation should be excised with adequate borders; this is particularly indicated if the nevus is of the junctional type. Malignant melanoma must be widely excised locally, and the resultant wound may require a skin graft; regional node dissection is recommended in addition to local excision whenever possible.

The prognosis is extremely guarded in all cases of malignant melanoma, as shown by the following statistics (from Ackerman and Del Regato): If distant metastases are present when the patient is first seen, the chances of five-year survival are nil; following dissection of nodes which are obviously positive on clinical examination, fewer than 5% survive 5 years; following dissection of nodes which are found to be positive microscopically, fewer than 10% survive 5 years; following dissection of nodes which are found to be negative microscopically, 30% survive 5 years.

CARCINOMA OF THE SKIN

Carcinoma of the skin is the most common form of cancer. It is most frequently seen in patients over 40 years of age, but may occur much earlier. Chronic exposure to sun and wind with development of keratotic lesions is a predisposing factor. Skin cancer is therefore most often noted on the face and dorsum of the hands in persons of fair or ruddy complexion.

Classification and Diagnosis.

A. Basal Cell Carcinomas: These may develop on keratotic lesions or on normal skin. They are frequently multicentric, sometimes pigmented, and are most often seen on the scalp, nose, nasolabial fold, eyelids, lips, chin, and forehead. Growth is by extension and infiltration, or may be exophytic, producing large, protruding tumors. Metastasis is rare.

Basal cell carcinomas are usually elevated, circumscribed, "pearly" lesions which later develop into an ulceration with rolled edges. The so-called "rodent ulcer" infiltrates and destroys widely and deeply, whereas the exophytic type develops large outgrowths with relatively little infiltration.

B. Epidermoid (or Squamous Cell) Carcinomas: These usually arise from keratotic lesions and are most frequently seen on the ears, the preauricular, temporal, and malar regions of the face, and on the dorsum of the hands. They may arise on unexposed areas of poor vascularity, such as in unhealed chronic burn scars and ulcerations, areas of radiodermatitis, and on chronic inflammatory lesions. These tumors may be multicentric and may metastasize to neighboring lymph nodes, although this does not often occur on the face. The percentage of metastasizing epidermoid carcinomas is about 5% on the face, 20% on the upper extremities, and 30% on the lower extremities.

Epidermoid carcinomas frequently begin as warty growths on preexisting keratoses which crust over, ulcerate, crust over again, and gradually spread with rolled indurated borders. Extension may be peripheral or deep, or both. Secondary infection is present after ulceration.

Treatment.

Many methods of therapy are in use, including electrocoagulation, escharotics (Mohs), roentgen or Curie therapy, and surgery. The objective of treatment is complete removal or destruction of the tumor, bearing in mind that the first opportunity is the best.

A. Surgical Excision: Complete excision, with microscopic study of the borders of the specimen, is recommended unless surgery is contraindicated. Excision must be wide and deep. Recurrences are due either to inadequate surgery or to multicentric lesions. In most instances, an immediate repair may be accomplished by direct approximation of the wound or by the use of local flaps. If the resultant wound is very extensive, or if a definitive repair is contraindicated because of the type or location of the tumor, temporary healing may be obtained by means of a split-skin graft.

Regional node dissection is undertaken at the same time when indicated.

B. Roentgen and Curie Therapy: Roentgen therapy is more commonly used than radium in the treatment of carcinoma of the skin because more efficient methods of application and control are available. A balance must be made between insufficient dosage, with recurrence, and overdosage, with resultant radiodermatitis. As in other forms of treatment, results vary according to the experience of the operator.

Radiated tissue is not suitable for use in any subsequent plastic procedure, and this must be borne in mind if later reconstruction is to be considered.

C. Chemosurgery: The Mohs technic of using escharotics under histopathologic control has a place in the treatment of extensive lesions with indefinite boundaries. This method should be reserved for those cases in which other forms of therapy cannot accomplish the same purpose. Reconstruction is undertaken after the tumor has been eradicated.

D. Electrocoagulation is not recommended, inasmuch as superior methods are available. The extent of tumor destruction cannot be accurately controlled with this technic, and recurrence is therefore not infrequent.

FACIAL WOUNDS

Immediate Treatment.

Primary repair is extremely important. Proper immediate care of facial wounds may eliminate the need for later revision, or, by conserving tissues, will facilitate any further work which proves necessary. Repair may usually be done 12-20 hours after the injury. Tetanus prophylaxis should be given as indicated (see p. 130).

A. Cleansing and Debridement: Preliminary cleansing with mild soap and water or pHisohex® is done prior to local anesthesia. The wound must then be thoroughly irrigated with saline, lifting skin flaps to expose pockets. All foreign bodies should be removed; dirt or grease ground into the tissues should be removed insofar as possible to minimize tattooing.

Ragged edges and obviously devitalized tissue should be excised However, one should be conservative in debriding tissues of the face because the excellent blood supply to this area reduces the threat of infection. Bone should be conserved even if it is detached. Portions of facial skin which have lost their blood supply may sometimes be converted to full-thickness grafts and used in the repair. Portions of the ears, nose, or eyelids, if not too badly traumatized, should be trimmed, gently washed in saline, and used in the repair; many times these will survive as composite grafts, and nothing is lost in the attempt. Suturing conforms to the principles described on p. 439. Drainage is usually not required. If there are abraded surfaces, a layer of petrolatum gauze may be applied to the wound. •A pressure dressing is then advantageous, with the pressure evenly distributed. Elastoplast® or a head wrap of Kerlix® gauze (or both) is often used for this type of pressure dressing.

B. Repair:
1. If no tissue is lost - Excise beveled edges to make right angles with the surface of the skin. It is not necessary to undermine the skin. If the suture line crosses a flexion crease which will produce webbing after healing, a small Z-plasty (see p. 460) may be done to avoid the necessity of later revision.

2. If tissue is lost - Avoid any distortion of features. Avoid tension on suture lines. Several procedures are available for repair of tissue-loss injuries.
   a. Undermining local areas will often provide sufficient tissue for the closure of wounds of moderate size.
   b. Trimming avulsed skin and using it as a free full-thickness graft.
   c. Use of local rotated or interpolated flaps.
   d. Full-thickness skin grafts taken from suitable areas.
   e. Split-thickness grafts may be used in large defects. These may be revised or replaced later if necessary.
   f. For losses involving cavities which cannot be immediately repaired (such as loss of a portion of the nose), suture the skin to the mucous membrane around the margin, wait for healing, and reconstruct later. Similarly, in loss of a portion of the ear, suture skin to skin. Always replace any avulsed parts of the facial features if the avulsed tissues are available and not too badly traumatized.

C. Postoperative Care of the Wound: Examine the wound after 48 hours and reapply a similar pressure dressing. Look for hematomas and any evidence of infection. If a hematoma is present, sufficient sutures should be removed so that the trapped blood can be expressed. If there is any evidence of infection, obtain culture and sensitivity studies and remove sufficient sutures so that drainage can take place. The appropriate antibiotic should be given in large doses and discontinued as soon as the infection is under control.

Remove sutures early. Half of the sutures may be removed on the third or fourth day and the remainder on the sixth day. The incision line should then be supported with adhesive butterflies for another 10 days.

Prognosis.
Prognosis depends on many factors, including the extent of the original injury, the method used in repair, and the inherent healing powers of the individual tissues. Time will tend to fade and soften scars, smooth contours, and blend grafts with surrounding tissues. An interval of 2 years should elapse before evaluating the final results.

FRACTURES OF FACIAL BONES

The most common types of fractures of the facial bones, in order of frequency, are fractures of the nose, mandible, malar, maxilla, and multiple fractures. All are caused by external force.

Edema may disguise or accentuate the deformity. Inspection and palpation are important guides to diagnosis. These should be supplemented by x-ray examination.

Displacement of bone fragments is determined by the direction of the fracturing force and by muscle pull, as seen in mandibular fractures, where the posterior fragment is displaced upward and the anterior fragment downward.

FRACTURES OF THE NOSE

If nasal fractures are seen early, before massive edema accumulates, they may be reduced at once. If treatment has been delayed it is better to wait until the swelling subsides so that more accurate diagnosis and reduction can be done.

Nasal fractures are usually reduced under local anesthesia, using 1% lidocaine (Xylocaine®) with 1:100,000 epinephrine solution, supplemented with 10% cocaine and epinephrine topically. General anesthesia (administered intratracheally) is preferable in some cases and is essential in children.

After anesthesia has been administered, clear the nose to establish an airway and aid in visualization of interior structures. Examine the septum for signs of hematoma, and incise and evacuate any clots present.

Types of Nasal Fractures and Treatment.
A. Simple Fractures: Gently disimpact the bones by internal manipulation with Walsham's forceps or a broad elevator. Align bones by a combination of internal pressure and external manipulation with the thumb and index finger.

Packing usually is not required unless persistent bleeding is a problem. In such a case the packing should not be disturbed for at least 3-4 days, since early removal will very likely cause renewed hemorrhage. Wait until the gauze is well saturated with secretions so that it may be removed easily. If a posterior pack is indicated, a No. 14 F. catheter may be inserted through the nostril and the 5 cc. bag inflated. The catheter is then fixed anteriorly with a bolster, and sufficient pressure applied (in combination with the anterior packing) to control bleeding. Although this method is simple and easy to apply, it is probably not as effective in some cases as posterior packing with gauze.

Once the bones have been disimpacted and replaced in normal position, there is no muscle pull which will displace them. Therefore, an external splint is required only for protection. This is left on for 4-5 days.

B. Compound Fractures: Associated lacerations of the nose should be treated at the same time as the fracture. If treatment cannot be given within the first 12-20 hours, however, it is wiser to wait for healing of the skin lacerations before reducing the fracture.

C. Comminuted Fractures: Additional fixation may be required in cases of severe comminution and flattening of the nasal bones. After reduction, a No. 24 stainless steel wire mattress suture may be passed through the fracture sites from 1 side of the nose to the other. This suture is twisted over large buttons or lead plates, which holds the nasal bones in their advanced position. Care must be taken to avoid pressure points on the skin.

MALAR AND ZYGOMATIC ARCH FRACTURES

The dense body of the malar bone is not readily fractured. Fractures and dislocations most often occur at articulations: the malar frontal suture, the malar maxillary suture along the floor of the orbit, and the zygomatic suture. The anterior wall of the antrum and the zygomatic arch are also frequently involved.

Fracture of the malar and zygomatic arch may cause depression or flatness of the cheek, limitation of motion of the mandible due to mechanical obstruction, diplopia due to displacement of the floor of the orbit, and emphysema due to laceration of the lining of the antrum. X-rays are of great value in diagnosis.

Treatment.

Reduction should be done early if possible, but at least within 2 weeks so that the bones may be readily mobilized. The following alternatives are available:

  A. Temporal approach (Gillies), by incision through the temporal fascia and introduction of an elevator under the zygomatic arch.
  B. Direct traction on the body of the malar bone by means of a forceps or towel clip.
  C. Elevation by the antral approach through a buccal incision.
  D. Direct wiring of fracture sites is frequently required when there is considerable displacement.

FRACTURE OF MANDIBLE

Fractures of the mandible most often involve the region of the mental foramen, the angle, and condyle. A fracture in the mental region is often associated with fracture of the angle or condyle on the opposite side.

Displacement is manifested by malocclusion, and depends upon the direction of the fracturing force and upon muscle pull, which tends to elevate the posterior fragment and depress the anterior fragment. Diagnosis depends upon the presence of some or all of the following signs and symptoms: pain and swelling, hemorrhage at the fracture site along the alveolus, crepitus, and displacement. For accurate determination of the degree of displacement and for detection of fractures without displacement, x-ray examination is essential.

Treatment.

Interdental wiring may be used alone or in combination with other methods, depending upon the circumstances. For simple fractures without displacement or those which may be realigned and maintained without difficulty, wiring will be all that is required. Immobilization of the mandible to the maxilla should be maintained for about 8 weeks.

If teeth are absent on 1 side, an acrylic bite plate may be provided which is attached by a bar to a cap splint on the teeth of the opposite side. The plate contains hooks for interdental fixation.

If the patient is edentulous, his dentures may be utilized after
they have been trimmed to fit without pressure points at the frac-
ture site. The lower denture is then immobilized by 4 circumfer-
ential wires inserted from below. The upper denture is maintained
in place by a wire encircling the zygomatic arch on each side (in-
serted from above and entering the mouth in the posterior alveolar
region) and by an additional wire through the nasal spine (inserted
orally). These wires are then twisted around loops provided in the
denture. The upper and lower dentures are then wired together.

Fracture of the condyle is usually treated by simple interdental
wiring. Even though the displacement is marked, the functional
result is usually quite satisfactory.

Isolated fracture of the coronoid is rare and does not affect
occlusion, although there may be considerable displacement of the
fragment by temporal muscle pull. Simple interdental wiring is
indicated.

Pin fixation is usually used for fractures of the body or angle
of the mandible when the displacement may be corrected by manipu-
lation but cannot be maintained by interdental wiring alone.

Open reduction is indicated in fractures with marked displace-
ment which cannot be reduced and maintained satisfactorily by other
methods. The intraoral approach may be used in fractures of the
body of the mandible. The extraoral approach is used for fractures
involving the ramus or angle. Two stainless steel wires are in-
serted through drill holes in each fragment, the bones aligned ac-
curately, and the wires twisted firmly. Interdental wiring is then
used for immobilization.

FRACTURES OF THE MAXILLA

Fractures of the maxilla vary from simple unilateral fracture
without displacement to complete separation of the entire maxilla.
They are frequently associated with severe intracranial injuries
and fractures of other facial bones. The care of such fractures
may present a serious and complicated problem.

The direction of displacement depends upon the degree and di-
rection of force. The entire maxilla may be displaced downward
(by complete detachment), upward into the nasal fossa, or directly
backward.

Treatment.
  A. First Aid: Provide an adequate airway, since massive edema
     and associated nasal fractures are often present. Tracheostomy
     may be necessary. Support the maxilla temporarily by exter-
     nal bandaging with muslin or elastic material slung beneath the
     chin and tied over the head, thus utilizing the lower jaw for
     partial immobilization.
  B. Fixation: In simple fractures involving 1 side only, the maxilla
     is realigned to correct occlusion and an arch bar is then fitted
     and wired to the teeth.
        In severe fractures with complete mobility of the maxilla, a
     combination of methods will be required. These will include
     wiring of the teeth, suspension to higher intact bones, and in-
     terdental wiring to the mandible. Associated fractures of the

mandible or other facial bones are cared for at the same time. The use of cumbersome head caps and traction is reserved for the few cases in which simpler methods are not sufficient.

C. **Feeding in Jaw Fractures:** It is not necessary to extract teeth in order to give fluids; a nasogastric tube may be used for the first few days if necessary. A high-protein supplementary formula is used. Foods may also be prepared in a blender for administration in the same way.

SCARS AND CONTRACTURES

HYPERTROPHIED SCARS AND KELOIDS

The hypertrophic scar is raised, red, and indurated. It is confined to the area of the wound. In time, usually after 6 months to 2 years, these scars tend to soften, flatten, and fade.

Overgrowth of connective tissue is frequently seen in third degree burns, especially if allowed to heal by granulation. It also may result from infection, tension, or inaccurate approximation of wound edges. It is more often seen when the scars run across normal tension lines. The anterior chest is particularly prone to overgrowth - so much so that benign lesions should not be removed from this area without due consideration.

It has been noted that the tendency to this scar formation may diminish after puberty.

True keloids, which are most often seen in Negroes, tend to invade surrounding tissue. They do not usually improve with time, and may attain tremendous proportions.

Treatment.
The best treatment is prophylactic, by paying close attention to the general principles of repair and by avoiding elective operations in dark-skinned patients in anatomic areas where this type of scar reaction is likely to develop. Prophylactic roentgen therapy may be given as soon as sutures are removed, although its value is debatable. Do not attempt to excise or revise a hypertrophied scar until all reaction has subsided, or it will very likely recur.

Treatment of the true keloid is often unsatisfactory, and recurrence is common. Excision and repair frequently result in a larger keloid. Any attempt at removal is a calculated risk.

CONTRACTURES OF THE NECK

These usually result from third degree burns or surgical incisions crossing flexion lines. Contracture is minimized by early grafting of burns, but some degree may develop from contraction of split grafts. The extent of contracture depends on the amount of skin loss; in extreme cases the chin is pulled down to the chest, with ectropion of the mouth and lower eyelids.

Scar tissue must be removed and replaced with normal skin. Minor degrees of contracture may be corrected by Z-plasty (see

p. 460). More severe cases usually require pedicle grafting.

Possible donor sites are the chest (bi-pedicle advancement flap), the acromiopectoral or deltoid areas, the back, or abdominal skin via the wrist.

Free grafts are sometimes used, and should be almost full thickness. Some degree of contraction may develop after healing with this type of graft.

CONTRACTURES OF THE AXILLA

Contractures of the axilla are most often seen following third degree burns, but may result from any type of wound involving skin loss in the axilla or from surgical incisions which cross the normal creases and create webbing.

Moderate contracture may be repaired by 2 plastic flaps if the surrounding tissue is not heavily scarred.

Severe contracture will require complete dissection of scar tissue, and if this is necessary the remaining defect will be quite large. Rotating a flap into this defect and grafting the donor site, when feasible, is superior to the use of a split-skin graft. If insufficient tissue is available for a rotated flap, a free graft will be necessary. Maximal mobilization of the shoulder joint should be attempted at surgery; however, a full range of motion is not always obtainable at the time due to muscle or tendon contracture, which may later yield to active use of the joint.

FACIAL PARALYSIS

Paralysis of the facial nerve involves the muscles of expression and the buccinator. The affected side is without expression, and the deformity is increased by the action of the muscles of the normal side. The mouth droops, the eye cannot close, and mastication is impaired. Speech is also affected.

Treatment.
  A. Nerve Repair: Some evidence of spontaneous regeneration should appear within 6 months or not at all. Reinnervation may be attempted by decompression, end-to-end anastomosis, nerve grafts, or anastomosis with hypoglossal or spinal accessory nerves. However, the results are usually disappointing.
  B. Mechanical Support:
    1. Muscle transplants include slips of the temporalis to the eyelids and of the masseter to the lips. These transplants may become fibrotic in time, losing some of their dynamic action and serving as static support only. The use of the masseter usually affords a fairly good result.
    2. Fascial slings - These may be used as static support alone or in combination with the masseter to provide some degree of muscle action. Fascial strips encircle the orbicularis, extending to the unparalyzed side to obtain contralateral pull. These strips are then attached to the masseter muscle or the temporalis fascia to slightly overcorrect the asym-
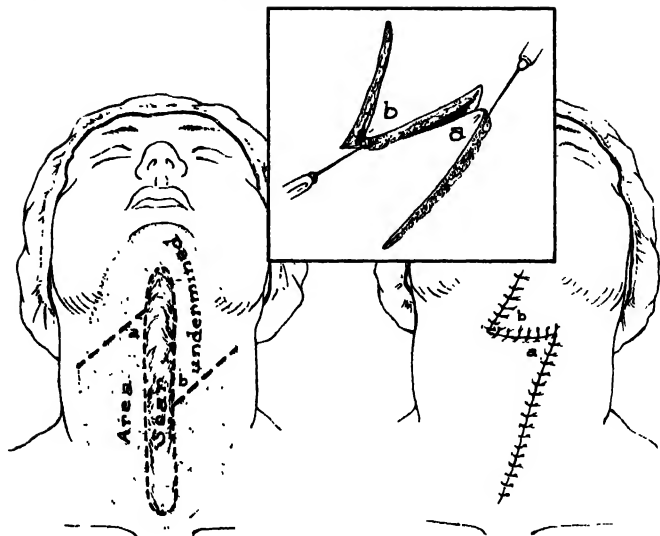
Repair of Anterior Neck Contracture by Z-Plasty. Left: Anterior neck contracture with incisions outlined for Z-plasty. Right: Transposition of flaps. Inset: Detail of flap transposition.
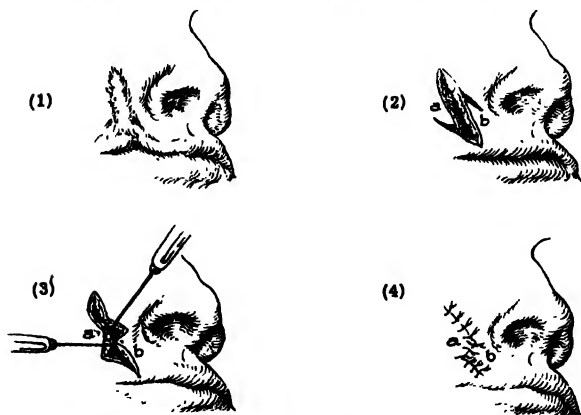
Repair of Scar Contracture of Lip and Cheek by Z-Plasty. (1) The defect. (2) Excision of scar and outline of Z-plasty. (3) Transposition of flaps. (4) Completed closure of wound.

metry. A sling may be inserted in some cases where return of function is ultimately expected to prevent stretching of muscles.
3. Rhytidectomy (face lift) removes sagging excess skin. Lateral tarsorrhaphy protects the cornea.
4. Sectioning of certain muscles of expression on the normal side is sometimes recommended to prevent gross distortion when the facial expression is animated.

COSMETIC PROCEDURES

RHINOPLASTY
(Plastic Surgery of the Nose)

The most common cosmetic defects of the nose are the long hump nose, the twisted nose, the deflected nose, and the saddle nose. Quite minor defects may for certain patients assume an exaggerated importance. An evaluation of the patient's motivation and personality is therefore an important part of the preoperative examination.

Preoperative photographs should always be taken.

The nose should be fully developed (16-18 years of age) before rhinoplasty is performed.

Surgical procedures consist of freeing the skin from the underlying bone and cartilage and altering the nasal framework as indicated, either by bone and cartilage removal or by grafting.

Incisions are closed with plain catgut, leaving adequate openings for drainage to avoid hematoma formation. Packing is lightly inserted to eliminate dead spaces. Adhesive strapping is carefully applied to mold the nose in its new contour; a molded splint of metal or modeling compound is then applied and fixed with adhesive.

Cold compresses over the eyes will help to reduce the edema which is sure to develop. This swelling and discoloration will gradually disappear during the first week

The packing is removed in 48 hours. Thereafter the nose is cleaned daily of crusts. The splint is maintained for 6-7 days.

The usual length of time before the patient resumes normal activities is 14 days.

RHYTIDECTOMY
(Meloplasty, "Face Lift")

Cosmetic surgery of the aging face is designed to remove redundant skin, thereby smoothing the face and neck and lifting sagging jowls. The "face lift" operation is a major surgical procedure which is not to be undertaken without complete physical and psychologic evaluation. The patient must be warned also that the aging processes cannot be halted by surgery, and that the tissues will continue to sag at a rate which cannot be predicted.

Incisions are placed so that the resultant scars are minimized.

Extensive undermining and freeing of facial and neck skin is neces-
sary to obtain a worthwhile result. Care must be taken to avoid in-
jury to the branches of the facial nerve or to the posterior auricular
nerve, and bleeding must be meticulously controlled.

After dissection has been completed and hemostasis is satis-
factory, the excess skin is pulled upward and excised. The skin
incisions are then closed with 00000 black silk and a pressure head
dressing is applied. Sutures are usually removed from the pre-
auricular and postauricular incisions on the fifth day; the sutures
within the hairline are allowed to remain for 8-10 days.

Edema and discoloration of the face and neck tissues is usually
moderate and will disappear in 7-10 days. Patients may ordinarily
return to their regular activities at the end of 2 weeks.

BLEPHAROPLASTY
(Correction of Baggy Eyelids)

Blepharoplasty is frequently done in combination with rhytidec-
tomy, but may be done separately. The upper eyelids will usually
require only the excision of excess skin, whereas the lower lids
may also require removal of periorbital fat which has herniated
through the muscle.

DERMABRASION

This procedure is used for the removal of superficial foreign
bodies resulting from road or industrial accidents, for improve-
ment of facial pitting following acne or chickenpox, and for removal
of superficial tattoos. The degree of improvement which can be
achieved with this technic will depend upon the depth of the defect.
In any pitted skin no more than 50% improvement should be antici-
pated. The procedure is contraindicated in Negroes or in any per-
son subject to keloid formation.

17 . . .

Hand Surgery

TRAUMA TO THE HAND

Any severe injury to the hand should be referred to a specialist in hand surgery if one is available. The objectives of management are to shorten the period of temporary disability during the healing stage and to minimize the degree of permanent disability where complete recovery cannot be anticipated. Successful management of the injured hand requires a clear conception of anatomy and a thorough appreciation of the principles of general surgery, orthopedics, plastic surgery, and neurosurgery. Care must be exercised not to interfere with the natural progress of healing and regeneration.

GENERAL APPRAISAL OF THE INJURED HAND

In hand injuries as in other fields of medicine and surgery, the initial evaluation by means of the history and physical examination is often the most important phase of total management.

History.
The answers to the following questions are of particular importance:
    (1) How did the injury occur? (Causative agent and mechanism of injury.)
    (2) When did it occur? (Time interval between injury and first medical attendance.)
    (3) What first aid care has been administered?
    (4) Is the wound clean or contaminated?
This information is of value in determining the extent and kind of damage, the degree of contamination which may be present, and the type of surgical and supportive treatment which should be given.

At this point it may be evident that specialist care is required.

In addition to specific matters relating to the injury, it is necessary to record pertinent collateral factors such as age, occupation, and associated systemic diseases (heart disease, diabetes mellitus, etc.). Right- or left-handedness should be noted since it may have important implications for the rehabilitation program.

Physical Examination.
Determine the type and degree of tissue damage and identify the structures involved. The actual wound is best not explored at this time. The general examination of the patient should not be overlooked.
  A. Type of Wound: Abrasion, laceration, puncture, crushing injury, burn, or combinations of these.

Wrist drop in
radial nerve injury

Radial Nerve

"Ape hand" deformity
in median nerve injury

Unopposed
thumb

Thenar atrophy

Median Nerve

Interosseus
atrophy

"Claw hand" deformity
in ulnar nerve injury
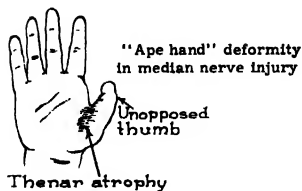
Ulnar Nerve

Testing Nerve Function and Sensation. Stippling shows sensory
distribution. Heavy stippling shows areas of isolated supply.
Heavy white arrows show the direction of movement in testing a
given muscle. Heavy black arrows show the direction of resis-
tance. The blocks show the site of application of resistance.

B. **Viability of Tissues Remaining:** Degree of circulatory impairment, hemorrhage, necrosis, infection.
C. **Tissues Involved:** In every hand injury, specifically test all tendons and nerves for function before giving treatment.
1. Gross deformity is caused by fractures and dislocations. X-ray studies should be ordered as indicated. X-rays will usually also show imbedded foreign bodies.
2. Loss of motion and abnormal posture and stance of the digits result from tendon damage.
3. Anesthesia (sensory function) and paralysis (motor function) are caused by nerve injury. (See p. 473.)

Accurate Records.

Good records are particularly important in hand injuries, where compensation or other medico-legal proceedings may require detailed written statements from the attending physician. The physician's record should include a diagram of the hand showing all wounds and skeletal deformities, notes on areas of loss of sensation, and notes on loss of motion of individual joints. It should close with his diagnosis and recommendations for treatment; an estimate of the duration of hospitalization, if any; and a prediction of the period of anticipated temporary disability and the degree of anticipated permanent disability.

MANAGEMENT OF THE INJURED HAND

Surgical Preparation for Hand Surgery.

The general principles which govern preparation for surgery are discussed in Chapter 2.

In undertaking the surgical repair of hand injuries it is well to remember that operating room facilities may be required even for seemingly minor procedures. Superficial lacerations may be sutured in the office only if it is certain that nerves or tendons have not been injured and that hemostasis will not be a problem. Strictly aseptic conditions must be maintained. Adequate assistance, a sufficient supply of the proper instruments, and good lighting must be available.

A stationary arm board should be used to immobilize the arm. A tourniquet is always necessary to provide a bloodless field and to keep the anesthetic in the extremity when using block or local anesthesia. A pneumatic tourniquet is preferred since it can be deflated for several minutes every 30-60 minutes (at least every 90 minutes) to verify the adequacy of circulation of the injured tissues, tó determine the adequacy of hemostasis, and to oxygenate the tissues if prolonged periods of ischemia are required. Before inflating the tourniquet (to 300-325 mm. Hg for an adult, 250 mm. Hg for a child, and 150 mm. Hg for an infant), the hand and forearm should be wrapped with elastic bandage or elevated to render the limb bloodless. In cases complicated by infection it is best not to wrap the limb with an Esmarch bandage (four-inch elastic rubber bandage), a snug Ace® bandage, or bias stockinet while the tourniquet is inflated; in these cases the part should simply be vertically elevated to permit blood to drain from the limb before the tourniquet is inflated. Wrapping an infected hand serves only to squeeze the pus into adjacent tissues.

Anesthesia.

General anesthesia is required in most severe hand injuries. Infiltration (block) with 2% lidocaine (Xylocaine®) around the major nerve trunks in the forearm is useful in many cases. Epinephrine is rarely necessary when an arm tourniquet is used.

Local block at the nerve trunks is often adequate. Injection of 5 or 6 ml. of 2% lidocaine around the ulnar nerve at the elbow and similar amounts around the median and radial nerves at the wrist affords satisfactory anesthesia. Adequate premedication is essential. Do not distend the nerve by injecting more than 1 ml. of solution directly into the nerve trunk.

Avoid brachial plexus and digital blocks and extensive infiltration of anesthetic locally into the hand. Brachial plexus blocks are rarely used since they are time-consuming, require expert technic, and are not without danger (i.e., pneumothorax, hematoma in the neck or chest, mediastinal infection, and injury to the nerve trunks themselves, resulting in temporary or permanent paresthesias and even paralysis). Digital blocks are dangerous since they produce local swelling of the soft tissues which may interfere with the circulation within the finger, often causing digital vessel thrombosis and considerable soft tissue necrosis. Epinephrine must never be used within a digit since it causes tissue necrosis and massive slough. Local infiltration of anesthetic into the hand tissues requires larger volumes of anesthetic solution, which has the disadvantage of distending the tissues and obliterating local landmarks.

Immediate Primary Repair.

In order to minimize the reaction to wound handling and operative trauma and to avoid spreading contamination, it is often desirable to resort to local cleansing and debridement of the wound and to close a clean wound with skin sutures, delaying the repair of deeper structures for several weeks. Factors in the decision to perform immediate primary repair of the deep structures include the time interval since injury, the degree of injury and contamination, the kind of emergency care which has been given, the general condition of the patient, and the surgeon's experience and the facilities available. A great deal can be gained by immediate primary repair: a single surgical procedure is often all that is necessary; temporary disability is lessened; and recovery of function is more satisfactory.

Primary repair is indicated in the following circumstances:

(1) If the interval since injury is short (preferably no more than 10-12 hours).

(2) If the degree of injury is slight. (In general, primary repair is all that is indicated for clean lacerations and the repair of deeper structures. In ragged crush wounds it is usually preferable to treat fractures and defer the management of tendon and nerve damage for secondary repair.)

(3) If contamination is minimal.

(4) If the patient's condition is good.

(5) If the surgeon's experience with hand problems is adequate.

(6) If proper facilities are available. (Operating room, tourniquet, good lighting, an assistant, and basic instruments: stainless steel sutures, Kirschner wire drill apparatus, retractors.)

Classification of Soft Tissue Injuries of the Hand

Type	Contamination*	Structures Involved	Interval for Primary Repair
Lacerations	"Tidy type"	Cut nerves and tendons.	Up to 12-18 hrs.
Puncture wounds		Cut soft tissues. Foreign bodies.	Up to 12-18 hrs.
Crushing wounds	"Untidy type"	Skin damage. Fractures.	Up to 8-12 hrs.
Avulsions		Loss of skin cover.	Up to 8-12 hrs.
Infected wounds		Tissue loss from slough.	Conservative ⅓
Amputations	"Untidy type"	Severed blood vessels.†	Up to 12-24 hrs.

*Any bacteria seen on a direct smear from the wound imply gross contamination. Such wounds must be treated conservatively.
†A digit will not survive if the neurovascular bundles have been cut.

General Surgical Principles.

A. Before Surgery: Protect wounds from further contamination and tissue injury until surgery can be performed under aseptic conditions. Avoid putting anything into the wound (instruments, sponges, antiseptics). Control bleeding with pressure dressings or a tourniquet (not clamps and ties). For these purposes it is often sufficient merely to cover the hand with voluminous light dressings, wrap with bias stockinet bandage, and splint with a cardboard or light yucca board splint, holding the wrist dorsiflexed with the digits in the position of grasp. Splint the hand in the position of function to maintain the optimum relation of soft tissue structures and bony fragments. This technic avoids contamination and further trauma, minimizes bleeding and pain, and prevents edema. Flat splints (e.g., tongue depressors) should not be used on the hand or digits.

B. In the Operating Room:

1. Cleansing - Protect the wound with sterile gauze, thoroughly wash the surrounding area with pHisoHex® or soap and water, and rinse copiously with water or normal saline solution. Then remove the gauze from the wound and wash the wound in the same way. Finally, wash the wound and surrounding area with benzalkonium chloride, 1:1000 aqueous solution.

2. Debridement - The objective of debridement is to transform a potentially contaminated, ragged wound into a clean surgical wound. Debridement must be meticulous and nontraumatic. Excise the wound edges and all ragged, grossly soiled, severely traumatized, or devitalized tissue, taking care not to sacrifice viable tissue or essential structures (skin, tendons, nerves, bones). Remove all devitalized and contaminated tissue, foreign bodies, and hematomas. Obtain hemostasis by ligation of major injured vessels.

3. Detailed inspection - Once debridement has been carried out it is possible to determine the amount of skin loss and the viability of the skin margins (circulatory adequacy) as well as the precise extent of injury to deeper structures. The cut ends of nerves and tendons, fractures, displaced bone fragments, and joint damage can be examined at this time and a definitive diagnosis of the injury established.

4. Exposure - Adequate surgical exposure is necessary to minimize trauma and to identify and repair damaged structures. **Caution:** Wound extensions and accessory incisions must be along the proper skin boulevards. It is imperative to avoid T-scars and wounds which may cause flexion contractures or jeopardize the circulation of surrounding tissues. One should not be hampered by a small wound or hesitate to make an auxiliary incision to locate a tendon end or give better exposure.

5. Hemostasis and drainage - Before the wound is closed, obvious bleeding vessels should be ligated with fine 000 or 0000 plain catgut ligatures (fine monofilament No. 40 stainless steel wire is sometimes used) and the tourniquet released to make certain that hemostasis is as complete as possible. Other bleeders should then be sought for, clamped, and ligated in such a way as to avoid trauma to adjacent tissues. (Minimize crushing of nearby tissues by clamping only small masses of tissue. In case of a generalized oozing it is advisable to elevate the limb and moderately compress the area of bleeding tissue; this generally suffices if no specific bleeders can be located.) Drains should be avoided because they may serve as a pathway of infection.

   NOTE: Through-and-through drainage is never used in hand surgery. When drainage is necessary, use narrow, short drains made of small rubber catheters with a safety pin in one end for ease of identification and removal. Rubber dam drains seal over within a few minutes, whereas catheter drains will permit drainage for 24 hours or more. They are used only in wet oozing wounds.

6. Methods of wound closure - Close the wound without tension on the skin edges. It is better to cover an elliptical wound with a thin split-skin graft than to close it under tension; or a full thickness local flap of skin can be advanced or rotated and the adjacent donor site covered with split-skin grafts. This is important not only in primary wound healing but also as a means of covering exposed deeper structures such as tendons, nerves, bones, and joints.

Dressings and Immobilization.

A. Dressings:

1. Ointments - Polymyxin-bacitracin (Neosporin®) ointment gauze, scarlet red ointment gauze, and hydrocortisone-neomycin (Neo-Cortef®) ointment are the anti-infective and anti-inflammatory dressings of choice since they are much less macerating to the tissues during healing. This advantage is due to the high melting point of the beeswax base used (in contrast to the usual petrolatum base).

2. Large protective bandages - Massive fluff dressings are applied (first separating the digits with thin single layers of flat gauze) and bound lightly in place without too much pressure with four-inch cotton, nonelastic Ace® bandage (avoid rubber elastic bandages) or stockinet bandage cut on the bias to increase its resiliency. The tip of at least 1 of the digits is left exposed if possible. Bandages are usually

extended to include the wrist and may be wrapped well up the forearm if the wrist is splinted.

B. Immobilization: Immobilize the hand and wrist in a light plaster splint in the **position of function** (i.e., the position of writing: wrist moderately dorsiflexed, proximal joints of the fingers flexed, fingers semiflexed, metacarpal arch curved, thumb in moderate opposition), or in the particular position necessitated by the nerve, tendon, or fracture repair (e.g., the wrist may be flexed to relax the repaired nerves and tendons at the wrist or in the palm, or the distal interphalangeal joint may be hyperextended in the correction of mallet finger). The position of nonfunction (straight or palmar-flexed wrist, straight fingers, or thumb at the side of the hand) must be avoided. Use flat splints of plaster crowning two-thirds of the circumference of the forearm, wrist, or digit (circular plaster dressings are rarely used). Leave the tips of the fingers exposed if possible so that adequacy of circulation can be verified during the immediate postoperative period.

Tetanus Immunization.

See p. 130.

Anti-infective Therapy.

If the wound is infected, a stained smear should be examined immediately and material taken for cultures and sensitivity tests. Prophylactic antimicrobial therapy should be reserved for grossly contaminated wounds or to increase the safe time factor in compound injuries or in anticipation of primary repair. In these instances it is imperative to take samples for bacteriologic diagnosis before antimicrobial drugs are administered.

The availability of anti-infective drugs is no justification for careless or inadequate debridement or rough handling of tissues.

General Postoperative Measures.

A. Elevation: Elevation is best accomplished by placing the splinted forearm and hand on a pillow; the overhead frame is less satisfactory because the constricting effect of the ties may impair circulation. Elevation and rest minimize pain and stiffness due to swelling (bleeding, edema) and promote healing by maintaining a nearly normal extracellular fluid balance.

B. Dressings: Avoid changing dressings unnecessarily, as this interferes with healing.

C. Active Exercise: (Passive physical therapy by means of passive stretching, forceful manipulation, and dependent hot soaks is of no value and may be harmful since it causes pain, swelling, stiffness, and irreparable damage to healing tissues.)

1. Immobilized parts - Voluntary active exercises should be started as soon as wound healing permits (usually on the tenth postoperative day in simple wounds). In tendon injuries, gentle exercise should be started in 3 weeks. In fracture cases, active motion should be delayed until there is clinical or x-ray evidence that healing is well under way. Adherence of tendons during healing of soft tissues or fractures can be prevented by minimal **supervised** voluntary exercise at ten-day intervals.

    2. Unimmobilized parts - Active use of unimmobilized parts should be started within a few days of the injury. Voluntary, gentle flexion and contraction of uninvolved digits 5-6 times every hour is sufficient at first.

D. Splinting: After the period of positive immobilization for healing, active exercises may be supplemented by the use of rubber bands and splints fitted with elastic bands to gently mobilize the involved joints (e.g., the "dynamic elastic band splints" of Bunnell, reported in J.Bone & Joint Surg. 28:732-6, 1946). This increases the range of passive joint motion gently, and slowly mobilizes a stiff digit.

E. Ambulation: Early ambulation may be permitted with the injured hand well elevated above the head or in a sling.

TENDON INJURIES

The diagnosis of tendon injury is made on the basis of loss of voluntary motion of a specific joint or of abnormal stance or position of the involved finger or finger segment.

The objective of repair is to reestablish the continuity of the tendon in order to restore voluntary motion and strength to the part. The gliding ability of the tendon should be maintained by handling the tendon ends gently and by preserving the tendon sheath or gliding surface.

When the time interval since injury is less than 12 hours and proper facilities are available, immediate primary repair of the soft tissues and involved deeper structures is indicated. However, if the operator lacks experience in nerve and tendon surgery it is better simply to treat the wound and close it with skin sutures and apply anti-infective dressings. The delayed primary tendon (and nerve) repair can then be carried out several weeks later after induration has subsided and the danger of latent infection has passed.

Early voluntary motion during healing and for 4-8 weeks after healing is essential to ultimate recovery of maximum function.

Extensor Tendons.

Diagnosis of extensor tendon rupture is made on the basis of loss of voluntary extension of a digit or 1 of its segments. Repair can usually be accomplished without additional exposure and using only a minimum of suture material. The wrist and finger are then splinted in extension for 4 weeks. Voluntary active exercise is started on removal of the splint. Restoration of useful range of motion is the rule.

A. Tendons severed in the forearm, on the dorsum of the hand, or at the base of the thumb are approximated with a buried braided, medium strength, stainless steel wire suture. A Swedish Fagersta® wire is preferable. Cut the ends off short so that the cut ends will lie along the tendon surface and not extend into the adjacent tissues.

B. For tendons severed on the dorsum of the thumb or fingers, a simple figure-of-eight suture or roll-type suture of monofilament No. 34 stainless steel wire generally suffices. These are left in place for 3 weeks and the hand, wrist, and involved finger joints splinted for 4 weeks on a volar plaster splint.

C. **Mallet Finger Deformity:** Rupture of the extensor tendon at the distal joint of a finger is usually treated with a Bunnell type suture: A single No. 34 monofilament, stainless steel, withdrawable wire suture placed with a pull-out loop proximally and fixed over the fingernail or over a button on the tip of the digit for 3 weeks.

Many surgeons are now treating mallet finger deformity by splinting the finger with a Kirschner wire placed longitudinally through the finger with the distal joint slightly hyperextended and the middle joint moderately flexed for 4 weeks. With this technic the encumbering external splinting is not required.

Flexor Tendons. (See p. 472.)

Division or rupture of the long flexor tendon of the thumb, the profundus flexors of the fingers, and the wrist flexors should be treated by primary suturing. The repair of a cut flexor tendon or tendons in the proximal segment of a finger presents considerable technical difficulty and should be undertaken only by a surgeon with special training in hand surgery.

Because flexor tendons generally retract to some degree, either proximally within the finger or even up into the center of the palm, additional exposure is required. Care should be taken not to cross flexion creases, and accessory incisions should be judiciously placed in the skin creases of the palm or longitudinally along the midlateral aspect of a digit.

The tendon sheath and its adjacent pulley should be slit longitudinally along the side for further exposure and to permit the repaired tendon to lie free during healing instead of within the limiting space of an intact sheath.

The tendon ends are best approximated nontraumatically with a minimum of mechanical handling; the use of stainless steel wire sutures minimizes rubbing or trauma to the surface of the tendon.

The wrist should be immobilized in moderate flexion with a dorsal plaster splint for 3-4 weeks. (The fingers should not be held flexed over a roll of gauze.)

Restoration of some degree of useful motion may be expected. Complete restoration of function is not possible because adhesions usually form within the digital sheath in flexor tendon injuries. Injuries in the proximal finger segments have the poorest prognosis.

A. **Forearm or Palm:** Tendon ends in the forearm or palm can be accurately approximated with buried braided wire sutures since there is sufficient soft tissue space to accommodate the tendon (which usually swells with edema, scarring, and trauma during healing). The wrist is then immobilized in moderate flexion for 4 weeks with a dorsal plaster splint. The fingers are left free. Gentle voluntary exercises may be carried out at five-day intervals under the surgeon's direct supervision for 3 weeks. After 3 weeks the patient may start gentle voluntary exercises several times every hour. Physical therapy is rarely given.

B. **Within the Digital Sheath of the Thumb and Fingers:** Only fair results can be obtained unless the following rather specialized technic is used: A single monofilament No. 34 stainless steel wire suture is placed nontraumatically in the proximal tendon end, advanced to the cut surface, passed longitudinally through the distal tendon, and fixed over a button on the tip of the finger.
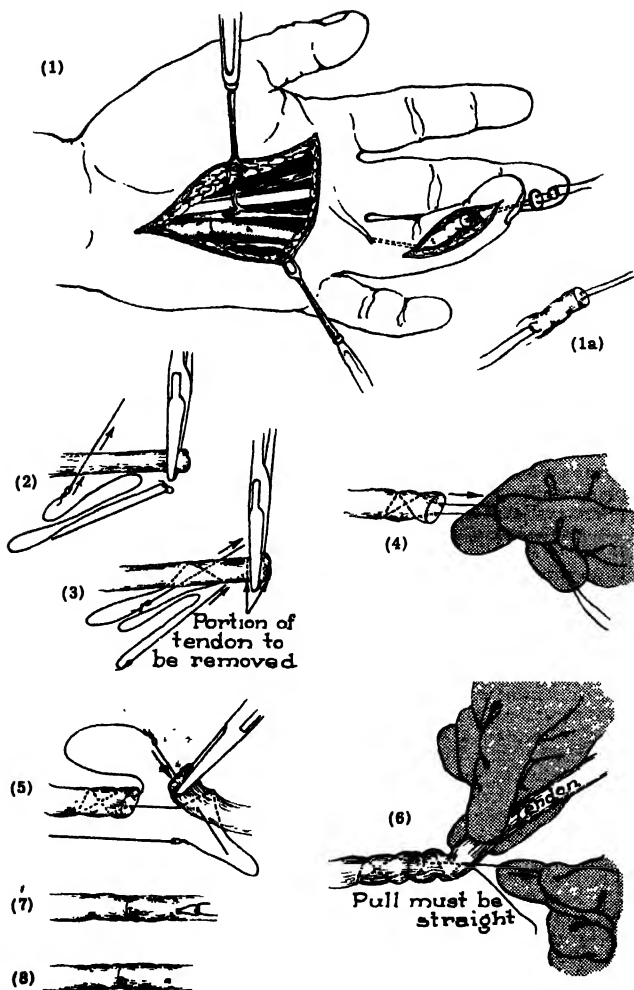
Methods of Flexor Tendon Repair. (1) Exposure and suture of flexor tendons. Within the finger, the pull-out method is used. In the palm, the buried suture technic is used. (1a) Enlarged view of suture placement in finger. (2, 3, 4) Details of suture placement in the proximal tendons. (5, 6, 7, 8) Details of suture placement in the distal end of tendon for buried suture.

A wire loop is placed in the proximal loop of the tendon suture so that the entire suture can be withdrawn in 3 weeks. This method has the advantage of requiring a minimum of suture material and affording a maximum of gliding surface.

C. Within the Proximal Segments of the Fingers: In this situation both the flexor profundus and flexor superficialis (sublimis) tendons are divided; uniformly poor results are obtained even in the best circumstances. The flexor tendon sheath and pulley must be unroofed and slit laterally well above and below the junction to prevent any constriction or adhesions and to provide as much space as possible for the healing tendon. The ends of the flexor superficialis tendon are removed. The distal end should be cut at the level of the middle joint to prevent a flexion contracture at this site from adhesions of the distal tendon stump within the proximal segment of the finger; the proximal end is cut off high so that it will be withdrawn proximally into the palm by the forearm muscle. Here the cut proximal end of the superficial flexor tendon rounds off within the tendon sheath in the palm. The withdrawable wire tendon suture technic is then used as described above.

The disadvantage of primary repair of the tendons within the proximal segment of the finger is adhesion formation at the tendon suture line, which causes permanent disability; for this reason primary grafts are sometimes recommended. If a primary graft is used, tendon junctures are at the distal phalanx (where adhesions serve the useful purpose of anchoring the graft in place) and in the center of the palm (where there is room for the juncture to swell so that adhesions are not likely to form). The graft ideally presents a gliding surface beneath the skin wound and theoretically moves freely at the site of trauma.

Most surgeons believe that grafting should be reserved for those cases in which primary tendon repair does not give satisfactory results. Thus it is considered preferable to suture the tendon primarily and to reserve the free tendon graft for a reconstructive procedure if further surgery is required.

NERVE DAMAGE

Any of the nerve trunks in the forearm and palm and along the volar aspects of the thumb or fingers up to the flexion creases of the distal joints are large enough to be found and repaired. The major nerve trunks in the forearm contain both sensory and motor components, and accurate repair is essential. Early repair will minimize atrophy of the small muscles of the hand and prevent trophic sensory changes in the digits.

Particular care must be taken to ensure proper rotation of the nerve trunk so that motor and sensory bundles will be accurately approximated at the suture line.

Diagnosis of nerve damage is made by testing for the presence or absence of sensitivity to cotton and pinpricks on the digits distal to the wound and by asking the patient to move his fingers. It is not necessary to examine the wound for cut nerve ends.
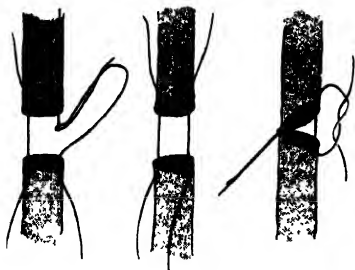
Placement of Initial Sutures in Nerve Sheath and Approximation of Nerve Ends
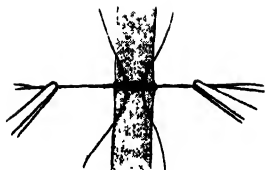
Placement of Additional Sutures in Nerve Sheath. Numbers indicate sequence of suture placement.

Incorrect (Left) and Correct (Right) Placement of Sutures

Blood vessels
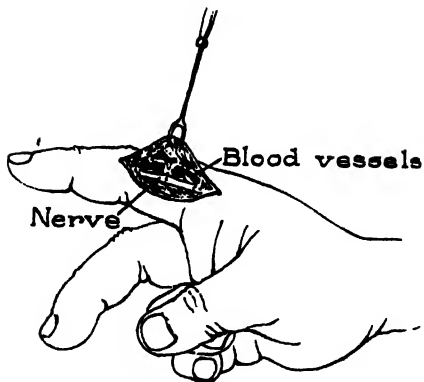
Nerve

Exposure Showing Sutured Digital Nerve

Technic of Nerve Suture. (See p. 474.)
  A. The cut ends of the nerve are identified, resected sufficiently
     to remove traumatized ends, and brought together with hand-
     sewn sutures. The ends of mixed nerve trunks should be
     marked with fine 0000000 black silk sheath sutures placed tem-
     porarily just above and below the cut surface to ensure correct
     rotation of the bundles when the 2 ends are sewn together.
  B. Nerve ends must be handled gently - never crushed or allowed
     to become dry. The traumatized ends should be resected
     several mm. This is particularly essential in crush injuries.
  C. The edge of the perineurium (not the bundles themselves) is
     then approximated with several simple interrupted sutures of
     0000000 black silk in the nerve sheath.
  D. Tension on the suture line is avoided by flexing the adjacent
     joints. This is essential where nerve substance has been lost
     as a result of a crush injury.
  E. The adjacent joints are immobilized immediately, and immobi-
     lization is maintained without interruption for at least 4 weeks.
     If loss of nerve substance has been such that the adjacent joints
     must be immobilized in flexion for 4 weeks, flexion should be
     reduced gradually (10-15° every 3-4 days) over the following
     2-3 weeks.

Prognosis.
     The prognosis for recovery of sensory nerve function is excel-
lent. Good recovery can be anticipated following repair of mixed
nerves if correct rotation is maintained at the suture line. Poor
results may be due to (1) inaccurate joining of the two ends, (2) in-
correct rotation of mixed nerve ends, (3) inadequate resection of
the traumatized ends, (4) tension (insufficient flexion of the adjacent
joints or extensive loss of nerve substance), or (5) inadequate or
improper postoperative splinting.

BURNS OF THE HANDS

     Small, superficial burns of the hands may be treated in the out-
patient department or in the office. Extensive burns should be
treated in a hospital, where aseptic technic can be controlled and
supportive measures instituted as indicated.
     See p. 17 for the general principles of the treatment of burns.
Those aspects of the management of burns which are specifically
related to burns of the hands are as follows:

Treatment.
  A. The eschar method is not used in the treatment of hand burns.
  B. Exposure Method: Exposure to promote early scab formation
     is used only in "island" burns, not for circumferential burns.
     The exposure method has the advantage of permitting exercise
     of the digits. The hand and wrist must be splinted and sup-
     ported in the position of function.
  C. Occlusive Dressing Method: This is the most satisfactory
     method for treatment of burns of the hand.
     1. The burn and surrounding areas must be thoroughly cleansed
        with soap and water (do not use chemical solutions) or

pHisoHex® under aseptic conditions. General anesthesia may be required. Devitalized tissue is then removed and a fine-mesh nylon (Owens cloth) dressing impregnated with neomycin ointment placed on the wound. Moderately snug pressure dressings are then applied and the hand is elevated on pillows. Dressings are changed infrequently (usually not oftener than every 3-4 days) under aseptic conditions to prevent secondary infection.

2. Early healing may be promoted by mechanical cleansing and Bunnell's solution* compresses after the first or second dressing on the third or sixth post-injury day. These dressings should be changed entirely every 24 hours.

3. If skin grafting is necessary it should be done early (within 14-21 days) to avoid exuberant granulation tissue and to promote early healing. Sterile Owens cloth treated with neomycin should be applied to granulated areas. Do not place skin graft margins in the line of push and pull.

4. Throughout the healing stage the part should be immobilized in the position of function (see p. 469). Early healing rather than early movement is the objective of treatment. Once healing is complete, however, gentle active exercises should be started.

5. Systemic chemotherapy should be given only as indicated by cultures and sensitivity tests.

Complications.
Restoration of complete function may be impossible following extensive or deep burns. Every effort should be made to minimize keloid formation and contractures. (Proper splinting lessens contracture.)

JOINT INJURIES

Contusions or crush injuries frequently result in sprains or dislocations of the joints of the digits. These injuries often do not receive the attention they deserve. The damage to the soft tissues around a digital joint may ultimately result in edema, joint stiffness, and persistent deformity sometimes even in the position of nonfunction.

Signs of sprain or dislocation include local tenderness, abnormal position or motion, swelling, and ecchymosis. Pain is prominent on active or passive motion or pressure on the extended finger. The diagnosis is based on the history, accurate examination, and x-ray studies.

Sprains.
A sprain represents an injury to a ligament of a joint or to the joint capsule. All simple finger sprains should be x-rayed. Because the extensive soft tissue injury around the joint requires a prolonged period of recovery, it is best to immobilize the injured joint with several layers of obliquely applied adhesive tape. A plastic splint holding the injured joint in a semiflexed position is

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*Bunnell's solution: Acetic acid, 0.5%, and glycerin, 20%, in benzalkonium chloride (Zephiran®), 1:2000 aqueous solution.

generally used.

The injection of local anesthetic into a joint is not recommended. The intra-articular injection of steroids is of value in sprains if prolonged disability is anticipated.

A. Thumb Sprains: Sprains of the thumb are generally accompanied by ligamentous rupture, as shown by partial luxation of a joint. Conservative treatment is generally preferred, using a nonpadded plaster hand cast. Surgical repair may be required for some of the more serious cases with ligamentous rupture and/or avulsing fracture, but this is generally a difficult and not too satisfactory procedure.

B. Finger Sprains: Crush injury producing overangulation of a finger joint may cause any degree of sprain, dislocation, or even an avulsion fracture. Splints should be applied promptly and maintained for 3 weeks. Immobilization on a curved splint relieves pain, permits prompt healing, and minimizes stiffness.

Dislocations.

Finger joint dislocations are associated with considerable soft tissue damage; treatment of the soft tissue wound should be delayed until x-ray studies are completed. Efficient immobilization is necessary throughout healing since early motion further traumatizes adjacent damaged soft tissues. The joint should be splinted for 3-4 weeks in a semiflexed position on a volar plastic gutter splint to minimize periarticular thickening, which generally persists for 3-4 months.

Reduction of a dislocation by traction along the axis of the digit and direct pressure over the joint is usually not difficult, especially if it can be done within a few hours following injury. (Occasionally it may be necessary to relax the flexor tendons by flexing the wrist.) Block anesthesia of the appropriate nerves of the forearm is recommended. Intra-articular injection of steroids is of value in reducing the soft tissue reaction (edema).

Dislocations (like sprains) are often associated with chip fractures. The prognosis is for prolonged disability and persistent deformity. Dislocation of the metacarpophalangeal joints of the digits is rare, but may be difficult to reduce if the metacarpal head buttonholes through the joint capsule; in this case surgical exploration may be necessary. Dislocations occur from tearing of the lateral and dorsolateral ligaments. Dislocations of the middle and distal phalanges are common, the distal phalanx displacing dorsally on the proximal one. In rare cases the head of the proximal bone may buttonhole through the anterior capsule of the joint, preventing reduction by closed methods.

Treatment.

A. Early Treatment of Sprains and Dislocations: Firm compression and elastic strapping within 1 hour after injury limits hemorrhage and edema. Immobilization and support on a plastic splint or in plaster, so that the torn ligaments are relaxed, is recommended for 3-4 weeks. Immobilization relieves pain and reflex swelling and interrupts the vicious cycle that stiffens the hand and causes osteoporosis, vasomotor changes, and prolonged disability.

B. **Treatment of Compound Injuries and Wounds of a Joint:** If the
patient is seen within 1-2 hours of injury, treatment consists
of thorough wound cleansing, meticulous debridement of the
joint, and copious irrigation with normal saline solution, fol-
lowed by primary soft tissue (joint capsule) closure, coverage
by swinging adjacent skin and soft tissue, and a split-skin graft
over the donor area. In these injuries one should give systemic
antibiotics to prevent infection. Absolute immobilization in the
position of function is required for 3 weeks (see p. 469). If
articular surfaces are exposed, the open joint must be con-
verted into a closed cavity. If possible, cover the joint with
local soft tissues and a rotational flap from adjacent skin; a
free skin graft may be used if necessary. Do not leave the
joint open or the surface exposed.

If the patient is seen too late for debridement and primary
closure, the capsule should be closed or the joint covered with
soft tissue and the wound packed open loosely.

Loose foreign bodies or bone and cartilage should be re-
moved. Collateral ligaments of the joint are best approximated
with a minimal amount of fine buried braided wire suture ma-
terial. If damage to articular surfaces has been extensive, if
the wound is grossly traumatized, or if a great deal of soft tis-
sue cover has been lost, primary arthrodesis is the procedure
of choice since this means of internal fixation with longitudinal
and oblique Kirschner wires least interferes with circulation
and does not require external splinting. The shortening caused
by joint resection minimizes the skin cover problems.

Crushing wounds generally are associated with skin loss.
Primary skin care is essential, covering the exposed joint
structure. Immobilization and support of the digits in the
position of function must also be effected. Avoid further ex-
posure of the bones and joints in compound crush injuries of
joints in a potentially contaminated wound.

FRACTURES OF THE HAND

General Principles of Management.

The general principles of fracture treatment outlined in Chapter
19 apply as well to fractures of the bones of the hand as to other
types of fracture. The following special aspects must also be borne
in mind:

A. **First Aid:** Immobilize and support the part to protect the in-
jured bony structures from further displacement and to prevent
additional damage to adjacent soft tissues. Make no attempt to
reduce fractures until skilled attention is available and an ac-
curate x-ray diagnosis has been established.

B. **Diagnosis:** Hand fractures are most commonly caused by crush-
ing injuries. Signs of fracture are gross deformity, swelling,
ecchymosis, and loss of function. Gentle palpation causes lo-
cal point tenderness. Gentle pressure along the axis of a long
bone causes discomfort at the fracture site. X-ray studies are
essential to accurate diagnosis.

C. **Anesthesia:** Inject 1% lidocaine (Xylocaine®) into the fracture
site or block the nerve trunks in the forearm with 2% lidocaine.

A pneumatic tourniquet is used to keep the anesthetic agent in the extremity. (Do not use epinephrine.) General anesthesia may be necessary to obtain adequate relaxation.

D. The normal bony framework of the hand and the position of function of the thumb and fingers (see p. 469) must be restored to permit healing, prevent deformity, and maintain normal tendon leverage and muscle balance. Care should be taken to support the wrist, the key joint in maintaining muscle balance of the long forearm and digital musculature.

E. Most hand fractures can be reduced by gentle manual traction, pressure, molding, and manipulation, and maintained by immobilization with a light form-fitting plaster casing on the dorsum and volar aspects. Avoid circular casing. Traction immobilization is seldom recommended. Do not use banjo traction. Unstable or compound fractures may require direct internal fixation by Kirschner wires. Open reduction is occasionally necessary.

F. The hand is prone to edema and stiffness. Do not immobilize any of the distal joints or uninjured adjacent digits unnecessarily. Do not bind an injured finger to an uninjured neighboring finger. Do not pack excessive dressings between the fingers.

G. The injured part must be sufficiently immobilized so that it will not move when the adjacent uninjured parts are moved. Immobilize in a nonpadded cast so that the injured part will be accessible for functional treatment of dislocations or fractures (Böhler). Do not use a flat board. Elevate the hand and forearm on pillows.

H. Voluntary active exercises should not be started until healing is well under way; early passive manipulation and stretching impede healing.

Classification and Treatment of Specific Types of Hand Fracture.

A. Simple Fracture Without Displacement: Finger tuft fractures due to crush injury, characterized by soft tissue swelling and subungual hematoma, are the commonest undisplaced finger fractures. Treatment consists of simple drainage of the hematoma under the nail, application of an antibiotic gauze dressing, and splinting of the finger in a semiflexed position on a volar plastic finger splint. Active motion can be started in 3 weeks.

B. Transverse or Linear Fractures of the Phalanges or Metacarpals (Usually Impacted): Treatment is by minimal immobilization in the position of function on a volar metal or plastic splint (or light plaster hand cast for metacarpal fractures) with the fingers in a semiflexed position. •

C. Simple Fractures With Displacement: A common type of fracture.

1. Most transverse fractures of the phalanges and metacarpals are displaced by the intrinsic muscle balance in the hand (see p. 480). These forces produce volar bowing of the phalanges (especially the proximal phalanges) and dorsal angulation of the metacarpals.

Treatment consists of accurate reduction and maintenance in the position of function. The Böhler frame splint is a satisfactory device for immobilizing the fracture after re-

Anatomy of Bones and Intrinsic Tendons
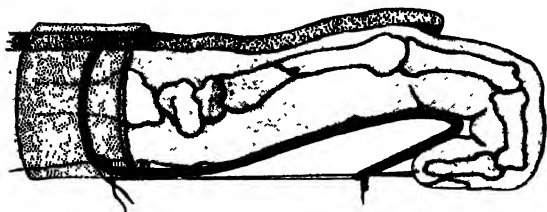
Metacarpal Fracture, Reduced on a Böhler Frame
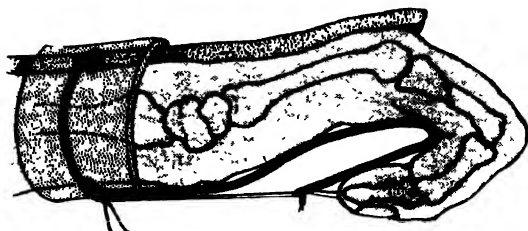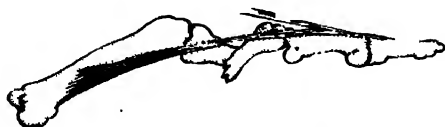
Fracture of the Proximal Phalanx, Reduced on a
Böhler Frame

duction by gentle traction and manipulation. Reduction is
then maintained by supporting the hand and wrist on a volar
plaster splint, with the wrist dorsiflexed and the injured
finger supported on a semiflexed outrigger wire frame in-
corporated into the plaster splint (see p. 480).

2. Oblique fractures with displacement are generally difficult
to maintain in reduction by closed methods; open reduction
and Kirschner wire fixation, preferably by a specialist,
may be necessary.

3. Chip fractures involving the joint surfaces present a diffi-
cult problem; even when accurate reduction can be achieved,
prolonged disability is to be expected.

4. Comminuted fractures are usually treated by closed manip-
ulation, molding the fragments into position on a Böhler
frame. Open reduction and Kirschner wire fixation may be
necessary. Care must be taken to avoid pressure on the
frame; to apply no more traction than is necessary to main-
tain reduction; and not to bend the finger and the frame at
the same time. Because of the prolonged temporary dis-
ability and the possibility of some permanent disability,
these fractures are best treated by a specialist in hand sur-
gery.

D. Compound Fractures: These are treated by early and accurate
open reduction through the wound and fixation with obliquely and
longitudinally placed Kirschner wires, with accurate reduction
and maintenance of position.

E. Later treatment of residual deformity may require osteotomy
and further Kirschner wire fixation, bone grafts, key grafts,
and chips.

THERAPEUTIC AMPUTATIONS

Removal of a portion of a digit may be necessitated by exten-
sive crush injury to the tissues or loss of adequate blood supply due
to any cause. The surgical objective is to preserve as much of the
digit as possible and yet provide satisfactory skin cover. The level
at which amputation is performed is determined by the degree of
damage, the age and occupation of the patient, and the digit involved.

Indications for Amputation.
Where prehension and sensation cannot be restored by recon-
structive procedures, a total or partial prosthesis is more useful
than a crippled hand or digit. In traumatic amputation of an entire
digit or where the digit is "hanging by a thread" or portions of
several digits have been detached, the amputated skin tissues may
be used as donor sites for skin grafts. For this purpose it is neces-
sary to excise a portion of the full thickness of skin, removing all
fatty tissue, so that the skin can be used as a free full-thickness
graft (Wolfe). This may be useful when the tip of a digit has been
sliced off, where one wishes to preserve as much as possible of
the stump, or when an exposed tendon, bone, or joint on an ad-
jacent digit requires immediate skin cover.

General Considerations.

Viable tissues should not be sacrificed. The objective is to restore maximum function with as good a cosmetic result as can be achieved. Provide a well-padded, nontender stump with adequate skin and soft tissue cover. Avoid tension in the closure and "dog ears" at the lateral borders of the stump. Shorten bone to accommodate soft tissue, and shape bone ends so that the stump will not be bulbous. Excise all nail matrix to avoid a small nail horn in the stump. Trim the digital nerves back to prevent local tenderness. Remove long tendons from the stump to avoid adherence to the stump or limitation of motion of the adjacent digits.

Technic of Amputation.

Amputation should be done only in a hospital. General anesthesia is usually required, although peripheral nerve block in the forearm can be used in selected cases. Use simple approximating stainless steel wire sutures. Massive postoperative fluff dressings provide sufficient immobilization. The hand should be kept elevated until healing is complete.

INFECTIONS OF THE HAND

Although minor infections of the hand are still frequently seen in clinical practice, the incidence of severe infections leading to permanent disability has been considerably reduced in recent years by greater emphasis on appropriate early care, increasing knowledge of surgical anatomy, and the availability of topical and systemic anti-infective agents.

The signs and symptoms of hand infections depend upon the tissue areas involved. The most common manifestations are pain, tenderness, swelling, immobility due to pain or swelling, and, at times, systemic symptoms of infection.

General Principles of Management.

Do not operate unless the surgical indications are clear. (No "look and see" surgery.)

A. Supportive Measures:
1. Elevation by means of pillows and an arm rest to ensure adequate circulation and drainage.
2. Bed rest with immobilization of the involved part.
3. Splinting, usually in the position of function. Types of splints vary with the part involved.
4. Antibiotic and chemotherapeutic agents - Establish etiology with stained smears and, when possible, culture and sensitivity tests (see p. 137).
5. Compresses - Lukewarm (not dependent hot soaks).
6. Treat any associated systemic disease.
B. Surgical Drainage: (See p. 483.) Antimicrobial therapy alone is usually not sufficient to combat a well-established pyogenic infection of the hand. Before drainage is attempted, be certain that pus is present and that the exact anatomic area and degree of infection are known. The patient should be hospitalized.

The operation (drainage) is performed under general anesthesia, using a pneumatic tourniquet. Great care must be exercised to avoid unnecessary incisions that will spread the infection or injure nerves, blood vessels, tendons, or tendon sheaths. The incision should be as long as the abscess to permit adequate drainage and packing, but it is important to avoid undermining or undercutting which might create pockets or deep sinuses. Loose wick packing is preferable to a tight pack because it permits freer drainage. The packing should be changed in 4 days and then daily thereafter so that healing may proceed gradually from the depths of the wound.

After operation, moist dressings of normal saline or Bunnell's solution (see p. 476) should be applied and the part splinted. The patient is then returned to bed rest and the arm elevated. Give systemic antibiotics chosen on the basis of sensitivity tests or a broad-spectrum antibiotic (see p. 613).

Immobilization should be maintained until the wound has healed. After pain and swelling have subsided and the wound has healed, the patient should be encouraged to perform voluntary exercises to prevent deformity and limitation of function and to restore full range of motion.

Common Sites of Pus in Finger

Removal of Nail for Paronychia, and Lateral Incision for Paronychia
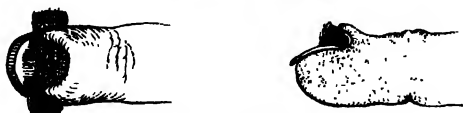
Packing of Drained Paronychia

**Line of Incision
for Dorsal Furuncle**

Incision for Drainage of Felon
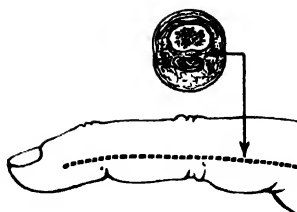
Incision for Drainage of Flexor Tendon Sheath Infection

Incision for Drainage of Infection of Dorsum of Finger
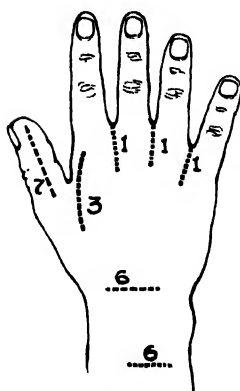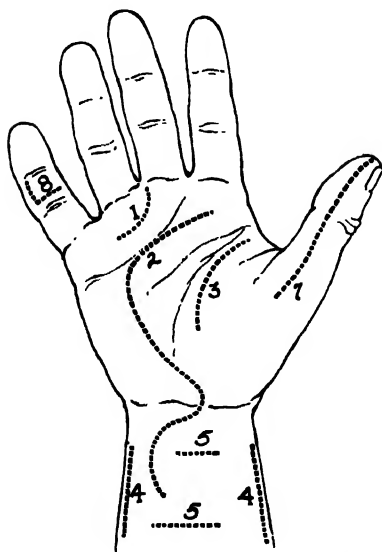
Incisions for Drainage of Palm and Volar Forearm (Left) and Dorsum of Hand and Wrist (Right). (1) Incisions for collar-button abscess of distal palm and dorsum of hand. (2) Incision in midpalm for midpalmar space abscess. (3) Incision in radial palm and dorsal thumb cleft for thenar space abscess. (4) Quadrilateral space incisions. (5) Exposure of proximal portion of flexor tendons. (6) Exposure of proximal portion of extensor tendons. (7) Exposure of tendon sheath of thumb. (8) Exposure of subcutaneous abscess of volar aspect of finger.

LOCALIZED PYOGENIC INFECTIONS

Puncture wounds, lacerations, or other trauma introduce skin contaminants beneath the surface of the skin, and localized infection results. Infections of this sort are usually staphylococcal, and become a collection of pus if the anatomic space is available. Diagnosis is seldom difficult.

Subepithelial Abscess.

Subepithelial abscess commonly occurs in the skin layers of the distal portion of the palm and on the volar aspect of the digits. It appears as a small, reddened swelling. The skin overlying the lesion or lesions should be cut away to permit free drainage. Care must be taken not to spread the infection by incising the deeper channels. No chemotherapy is needed.

Subcutaneous and Web Space ("Collar Button") Abscesses.

Localized swelling and pain are the principal clinical manifestations. Abscesses appearing on the volar aspect of a middle phalanx may invade the flexor tendon sheath. Abscesses of a proximal segment may extend into the interdigital web spaces, causing the fingers to spread apart, or into the palmar spaces, flexor tendon sheaths, or along the lumbrical canals. Treatment is by incision and drainage. No chemotherapy is needed.

A. "Collar button" abscess (so-called because the bones of the hand limit extension of the abscess along a narrow column or sinus which then swells above and below in the general shape of a collar button) in the interdigital cleft causes considerable tissue reaction, swelling, and pain. Treatment consists of drainage in the distal palm through a curved incision between the neurovascular bundles of the adjacent fingers, and a longitudinal incision on the dorsum followed by insertion of light packs in each wound. Do not use through-and-through rubber dam drainage.

B. Abscess on the volar aspect of a middle phalanx is treated by midlateral incision, wide enough to permit good drainage.

C. Proximal segment abscess is treated by a longitudinal midlateral finger incision curving into the distal palm. A light pack of loose wicking is inserted.

Folliculitis (Furuncle).

Furuncles on the dorsal aspects of the digits and hands in the hair-bearing areas are subcutaneous abscesses which begin in a hair follicle and spread locally in the subcutaneous space. Treatment consists of splinting and elevation and moist wet dressings. Incision should be avoided except for the purpose of removing pus or slough. No chemotherapy is usually needed.

Paronychia.

Paronychia is an acute abscess around the base of a nail which detaches the nail from its bed so that it acts thereafter as a foreign body. Chronic paronychia is commonly a dermatologic problem which is best treated by antibiotic ointment, general hand hygiene, protection from further trauma, and, in selected cases, removal of the marginal portion of the fingernail.

Elevate the eponychium and remove the proximal third of the nail. Compress or pack lightly under the nail fold until epithelization occurs (usually in about 2 weeks).

Felon.

Felon is a closed-space abscess of the pulp of the distal segment of a digit which results from spread of a paronychia or a puncture wound on the volar aspect of the digit. Swelling of the entire pulp of the digit due to severe soft tissue trauma causes extreme throbbing pain.

Surgical drainage is usually necessary. Make a dorsolateral hockey stick or fishmouth incision in the tip of the finger close to the nail and divide all of the vertical fibrous septa from the skin to the underlying phalanx, laying open the entire pulp space of the fingertip.

Care must be exercised to avoid the flexor tendon sheath and periosteum. Pack and leave open for four days, and apply lukewarm (107° F.) Bunnell's solution compresses (see p. 476) every 4 hours. The part should be kept splinted and elevated at all times until healing is complete.

Puncture Wounds.

Puncture wounds cause subcutaneous abscesses which may spread to the tendon sheaths or into the joints. Human bites are a common source of the infection. Apply warm moist dressings and give prophylactic broad-spectrum antibiotics as indicated. Splinting in the position of function is essential.

INFECTIONS SPREADING BY TENDON SHEATHS AND FASCIAL TISSUES

The spread of infection within the digits of the hand is along well-defined anatomic pathways. Anatomic familiarity with the flexor tendon sheaths and fascial spaces in the hands is important in the diagnosis and treatment of infection.

Acute Suppurative Tenosynovitis.

Acute suppurative tenosynovitis is a rapidly spreading infection of the flexor tendon sheaths which may be caused by a puncture wound or laceration on the volar aspect of the finger. The cardinal signs are as follows: (1) Slightly flexed position of the digit; (2) uniform swelling along the volar aspect of the digit; (3) pain on extension of the digit; and (4) tenderness along the entire digital sheath.

Early diagnosis has important implications for therapy, since appropriate drainage should be started without delay. Differentiation from extensive subcutaneous space infection may be difficult, as some of the cardinal signs of tenosynovitis may be absent if the wound is open. Complications include spread into thenar and midpalmar spaces, the radial or ulnar bursae, the flexor tendon sheaths of the thumb and finger, the quadrilateral spaces of the forearm, and the digital joint spaces.

  A. General Measures: Absolute bed rest in a hospital with the arm and hand immobilized and elevated is an essential preliminary to early surgical drainage. Give supportive systemic therapy as indicated.

B. Surgical Measures: Make a midlateral incision and pack the entire sheath loosely with gauze wick. Avoid opening the interphalangeal joint capsules or injuring the digital neurovascular bundles. Apply massive compresses and return the patient to bed rest with the arm elevated and splinted.

Thenar Space Abscess.

Thenar space abscess may occur by direct extension from wounds of the palm or indirectly following rupture of a tenosynovitis or by extension from the radial bursae or the web spaces of the thumb or index finger. There is localized swelling and induration, tenderness, slight pain upon finger motion, loss of concavity of the thumb web, and dorsal swelling of the hand. Specific bacteriologic and anatomic diagnosis is required.

A. General Measures: Hospitalization, absolute bed rest, elevation, supportive systemic care, and systemic antibiotics.

B. Surgical Measures: Treatment is by multiple incision and drainage of the dorsal and volar aspects of the thumb along the cleft in the skin creases parallel to the metacarpal. (Avoid the first dorsal interosseous muscle, thenar motor nerve, and sensory digital nerves in the palm.) Loose wick packing should be used and a loose dressing applied. The patient should then be placed at bed rest with the arm elevated and splinted. Frequent wet compresses should be applied and the wound packing changed daily after the fourth postoperative day. Dry dressings or ointment gauze may be substituted for wet dressings as the drainage subsides and the wound edges close in.

Midpalmar Space Abscess.

These may occur as direct extensions from wounds of the palm or as indirect extensions following rupture of an abscess in the ulnar bursa, tenosynovitis of the third, fourth, and fifth digits, or from adjacent web spaces. Clinical manifestations are identical with those of thenar space abscess except that the ulnar half of the palm is involved. (The third metacarpal is the watershed.)

General care is similar to that given for thenar space abscess. Surgical treatment consists of making a curved palmar incision and inserting a loose gauze wick. A loose dressing is applied and the patient placed at bed rest with the arm elevated and splinted. Apply wet compresses until the wound granulates and appears to be closing in without deep pockets.

Dorsal Subaponeurotic Space Abscess.

Abscess of the space beneath the tendons on the dorsum of the hand may occur as a result of direct wound infection or, rarely, rupture of a dorsal tenosynovitis. There is considerable swelling, tenderness, and pain on movement of the fingers.

General care is the same as that given for thenar space abscess. Incisions are made longitudinally between the extensor tendons on the dorsum of the hand (taking care to avoid the extensor tendons and the dorsal branch of the ulnar nerve), and loose drains are inserted.

Abscess of the Quadrilateral Space of the Forearm.

This may occur by extension from a midpalmar space abscess or by rupture of a radial or ulnar bursa infection. It is characterized by deep swelling, induration, and pain in the distal third of the volar aspect of the forearm.

General care is as for thenar space abscess. Make incisions in both lateral aspects of the wrist region and insert wick or short rubber catheter drains.

CHRONIC NONPYOGENIC INFECTIONS

Tuberculous Tenosynovitis.

Tuberculous tenosynovitis is manifested by boggy swelling in the palm and the distal volar forearm, associated with mild pain and some limitation of digital motion. It is quite rare. Treatment is by complete excision of the involved sheaths and spaces, followed by primary closure, splinting of the wrist in the neutral position, systemic antituberculosis chemotherapy, and months of rest.

Tuberculous Osteomyelitis (Dactylitis).

Treatment consists of prolonged splinting of the hand and finger in the position of function, chemotherapy, and general supportive care. In severe or advanced cases resection of the joint or amputation of the involved digit is recommended.

MISCELLANEOUS DISORDERS

Trigger Finger.

Trigger finger is caused by tension on the flexor pulley at the base of the thumb or finger. Hydrocortisone injections are indicated in mild cases where the edema and tissue reaction are of short duration. In severe or chronic cases the pulley should be unroofed surgically.

Carpal Tunnel Syndrome.

This is caused by narrowing of the carpal tunnel or compression of the space, with associated median nerve neuritis.

Dupuytren's Contracture.

Thickening of the palmar fascia in varying degrees results in a contracture of the digits. Slow progression is the rule. Surgical fasciectomy is required for cure, but considerable improvement in function can be afforded by local subcutaneous fasciotomy. This is recommended in elderly poor-risk patients, or where the contracture is caused by a single band of fascia across the palm in the region of the metacarpophalangeal joint.

Ganglion.

A ganglion is a cystic tumor which most frequently occurs around the wrist joint but may also be seen at the proximal interphalangeal joint of the fingers and on the flexor pulley at the base of a finger. It arises from degeneration of the connective tissue of the joint capsule, and is generally associated with local trauma. Swelling causes mechanical interference with function. Mild subjective symptoms are usually present, e.g., weakness of grip.

If a ganglion is large enough to interfere with function or if symptoms are severe, treatment is by surgical excision with wide removal of the base of the lesion.

Rheumatoid Tenosynovitis.

Rheumatoid tenosynovitis is characterized by boggy swelling in the palm and distal volar forearm, with considerable local pain and discomfort on active or passive motion of the digits. Other manifestations of rheumatic arthritis are usually present (dorsal knuckle pads, especially on the proximal interphalangeal joints; trigger fingers, carpal tunnel syndrome, and de Quervain's tenosynovitis of the abductor tendon sheath of the thumb).

Mild or early cases are treated conservatively with splinting, elevation, rest, mild heat, and local injections of hydrocortisone in the tendon sheath. Severe or chronic cases require surgical excision of the proliferative material around the lesion.

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Neurosurgery

GENERAL PRINCIPLES

A detailed history and thorough neurologic examination are the most important steps in arriving at a diagnosis and selecting the proper therapy in neurologic diseases. Special attention by direct and/or leading questions should be given to headache, visual difficulties, episodes of loss of consciousness, impairment of higher intellectual functions, weakness, incoordination, sensory disturbances, etc. The chronology of the history may reveal a steady progression or fluctuation of symptoms.

Ophthalmoscopic examination is an important part of the neurologic survey. It is usually unwise to use mydriatics, as the size of the pupils and their reaction to light are valuable signs of changes in intracranial pressure. If a mydriatic must be given for better visualization of the retina, a note to this effect should be made in the patient's chart so that subsequent examiners will know the cause of the pupillary changes. Never artificially dilate the pupils in head injuries.

History taking and neurologic examination are designed to answer 2 questions: (1) Where is the lesion? and (2) What type of lesion is it? Additional diagnostic studies are usually required for definitive diagnosis.

Skull X-rays.

In 50% of cases the presence of a brain tumor can be inferred by examination of plain films of the skull. The plain film is therefore a most important preliminary study. At least 5 views should be taken: anteroposterior, posteroanterior, right and left lateral, and a half-axial view to visualize the area about the foramen magnum. Stereoscopic views are helpful also. Skull x-rays should be taken in all cases of significant head injury to visualize fractures, depressed bone, foreign bodies, pineal shift, etc., and for medico-legal purposes.

Spine X-rays.

' Anteroposterior, lateral, and right and left oblique films should be taken of the appropriate spinal region when there is clinical indication of injury or disease involving the vertebrae and/or spinal cord. Tumors and other space-occupying lesions may be shown by foraminal enlargement, increase of interpedicular distance, erosion or osteoblastic changes, or calcification.

Electroencephalography.

EEG's are indicated in the following circumstances: (1) When there are episodes of alteration in consciousness, to aid in the differentiation of convulsive seizures from syncope, and in carotid

sinus syndrome. (2) Following serious head injuries, serial examination has considerable prognostic value. (3) When the presence of a focal cerebral lesion is suspected, serial examination may be helpful.

Lumbar Puncture.

Lumbar puncture is used to obtain information concerning 1 or all of the following: (1) Intracranial pressure, (2) CSF protein, cells, glucose, and chlorides, (3) serology and colloidal gold, (4) evidence of subarachnoid hemorrhage, and (5) culture, smears, and agglutination reactions. In general, the following information should be obtained in every lumbar puncture: (1) Initial pressure. (2) Gross appearance: 1 ml. of CSF is placed in each of 3 test tubes and the color and clarity of the first tube is compared with that in the last tube. A cell count should be done on the last tube. (3) Final pressure. **Note:** The Queckenstedt test should be carried out only in cases with suspected space-consuming lesions in the spinal canal and never in patients with suspected intracranial lesions.

A. Contraindications:

1. Increase in CSF pressure (as determined by the presence of papilledema) - The danger in this situation is that the reduction of pressure occasioned by the escape of CSF at the time of puncture or after the needle is withdrawn may produce (1) herniation of the medial-inferior temporal lobes into the incisura tentorii or (2) herniation of the cerebellar tonsils into the foramen magnum. Either may cause serious complications or death due to sudden compression of vital brain stem structures.

2. In cases where spinal cord tumor is suspected, lumbar puncture should be done only when all arrangements have been made to perform a myelogram if a total manometric block is found. If this is not done, all available fluid below the spinal fluid block may be inadvertently drained off, in which case the crucial myelogram required later may be impossible to obtain.

B. Technic: Lumbar puncture may be done with the patient in the sitting position but is usually carried out as follows:

1. The patient is placed on his side with his legs, thighs, and neck flexed so that the spinal column is well "bowed out" toward the operator.

2. The skin is suitably prepared with an antiseptic solution and infiltrated with a local anesthetic.

3. A No. 20 lumbar puncture needle is introduced between the spinous processes of the selected interspace (L3-L4). •The needle must be perpendicular to the skin. As the needle is advanced through the interspinous ligament, one can usually feel a "pop" when the needle point penetrates the ligamentum flavum; at this point the stilet should be partially withdrawn to see if CSF appears. If not, the stilet is reintroduced, the needle gently advanced a few mm., and the stilet again partially withdrawn until CSF is obtained. The stilet is then immediately replaced to prevent loss of CSF.

4. The stilet should never be completely withdrawn, since loss of CSF may be extremely dangerous in the presence of increased intracranial pressure or spinal cord tumor. A

three-way stopcock adjusted to prevent flow is carried in the left hand and the stilet slowly removed with the right hand so that the stopcock can be immediately inserted in the needle hub to prevent loss of fluid. (An alternative method is to remove the stilet when the ligamentum flavum has been penetrated and to attach the stopcock and water manometer to the needle. As the needle is advanced and the arachnoid penetrated, the fluid pressure can immediately be determined and fluid loss and sudden changes in pressure prevented.)

5. The patient should slowly straighten his legs and neck so that intracranial pressure can be measured accurately. Flexion of the legs will compress the abdomen, which in turn may raise the intraspinal pressure, and flexion of the neck may compromise the venous return in the jugular system and also produce rises in pressure. If a spinal block is suspected, the Queckenstedt test should be done at this point by recording the pressure again after an assistant has compressed both jugular veins for 10 seconds. After release of jugular compression the pressure should be recorded at ten-second intervals for 30 seconds. With normal dynamics the pressure should promptly rise and fall. With a complete block there will be no change. Partial block causes an incomplete and slow rise and fall.

6. After measurement of CSF pressure as above, 1 ml. samples should be collected in each of 3 test tubes drop by drop under manometric control. If there is more blood in the first tube than the third, the blood is due to traumatic spinal tap, whereas in subarachnoid hemorrhage the fluid in all 3 tubes will be the same color.

7. The final pressure is recorded, the needle withdrawn, and a small dressing applied. The patient should remain flat in bed for 12-24 hours.

8. The cell count should be done immediately on tube No. 3 and the remaining specimens sent to the laboratory for pertinent chemical analyses.

Other Tests.

The information obtained from the history, neurologic examination, and the above-mentioned diagnostic tests is then used to determine what additional diagnostic tests may be required, e.g., pneumoencephalography, cerebral angiography, and ventriculography.

SPECIAL FEATURES IN MANAGEMENT

Neurosurgical patients frequently present the most difficult and involved management problems, both preoperatively and postoperatively. In many cases adequate management requires the attending surgeon's personal observation and evaluation of the patient as often as every 2 hours or even continuously. Sudden changes in intracranial pressure may produce an emergency situation in a matter of minutes or hours. Seizures or even status epilepticus may occur suddenly. Dilatation of a pupil, increasing obtundation, or progressive hemiparesis may develop in a matter of minutes.

Increased Intracranial Pressure.

This is a common and critical problem in neurosurgery. It will always be a complication in expanding intracranial lesions if sufficient time elapses before the patient seeks medical attention. The brain can compensate for a surprising amount of distortion which comes on slowly, but decompensation can occur swiftly if rapid changes in intracranial pressure occur. The patient may become unresponsive and moribund in a matter of minutes.

Increased intracranial pressure may be caused by (1) cellular and/or vascular injury or disease, producing cerebral swelling (so-called "cerebral edema"); (2) space-occupying lesions (e.g., neoplasms, hematomas, abscesses); (3) obstructions of ventricular and CSF pathways; or (4) interference with venous drainage from the intracranial cavity (e.g., tumor encroachment, thrombosis of dural sinuses), and (5) miscellaneous infrequent causes.

A. Symptoms and Signs: Headache, alteration in consciousness (from slight decrease in higher intellectual functions to deep coma), nausea and vomiting, papilledema (which may lead to blindness), extraocular muscle palsies (abducens: medial deviation of the eye; oculomotor: dilatation of a pupil may be a sign of transtentorial herniation of the temporal lobe).

B. Treatment:

1. Preoperative and postoperative - Restrict fluid intake. The intravenous administration of 100-300 ml. of 30% urea (90 Gm. in 300 ml.) over a period of 30-60 minutes may be valuable.

2. During surgery the most effective means of combatting increased intracranial pressure is by surgical removal of the causative lesion as completely and rapidly as possible. If total removal is not possible, as complete as possible subtotal removal (internal decompression) is indicated. If the intracranial pressure continues to be obviously increased after surgical treatment of the causative lesion, external decompression can be done by leaving the dura mater partially or completely open and by removing part or all of the bone flap.

3. Constant ventricular drainage by means of an inlying ventricular catheter is sometimes useful as a temporary measure to reduce increased intracranial pressure due to CSF block.

Seizures.

Aura, onset, type of seizure, and postictal state may provide significant diagnostic clues to the localization of pathology. A major seizure may be the precipitating factor causing sudden deterioration in a patient with increased intracranial pressure (see above). Postoperative seizures indicate either localized cerebral edema from surgical trauma, or a hematoma. If an extradural, subdural, or intracerebral hematoma is suspected, immediate reoperation is mandatory.

Medical treatment consists of anticonvulsant drugs in a dosage sufficient to control seizures. The usual regimen is diphenylhydantoin (Dilantin®), 0.1 Gm. (1½ gr.) every 4-6 hours orally, or 0.15 Gm. (2½ gr.) I.V. Anticonvulsants may be given alone or in combination with intravenous barbiturates.

Hypothermia.

This is a valuable adjunct in surgery to reduce the risk of operation upon extremely vascular brain tumors, arteriovenous malformations, aneurysms, etc. At currently used hypothermic temperatures (28-30° C.), the metabolic requirements of the brain are lowered sufficiently so that temporary occlusion of the entire arterial supply to the brain is possible. This affords the surgeon an opportunity to deal with these formidable lesions in a relatively dry field.

Other Supportive and Symptomatic Measures.

Because of impaired consciousness, paresis, etc., neurosurgical patients may be unable to take care of their own bodily needs.

A. Airway Obstruction: If the level of consciousness is severely depressed, frequent tracheopharyngeal aspiration is required to maintain an adequate airway. Tracheostomy should be done as soon as obstruction of the airway is imminent (see p. 166).

B. Urinary Retention: Continuous indwelling catheter drainage is usually required. The catheter should be changed every 7 days and urinary output maintained at a minimum of 1500 ml./day if renal function is normal. Prolonged use of an inlying catheter almost invariably causes a lower urinary tract infection, but antibiotic therapy should be withheld until systemic symptoms appear.

C. Decubitus Ulcers: The comatose, paraplegic, or quadriplegic patient is extremely susceptible to the formation of decubitus ulcers (sacral, over the greater trochanters, heels, elbows, etc.). These can be prevented by proper nursing care (see p. 64).

D. Restlessness: Restlessness is a frequent problem, particularly in patients with severe head injuries, and a search should be made for an obvious cause such as a distended bladder, blood in the CSF, hypoxia, etc. The most effective medications are small doses of chloral hydrate, paraldehyde, and the ataraxic drugs. NEVER USE NARCOTICS, PARTICULARLY IN PATIENTS WITH HEAD INJURIES.

E. Fluid Balance: Fluid intake should be sufficient to ensure adequate urinary output. Fluid balance and requirements in the neurosurgical patient are no different than in other areas of surgery, but certain dangers are peculiar to the neurosurgical patient:

1. Production or accentuation of cerebral edema by overhydration, with resultant hyponatremia, may cause increasing stupor and mimic postoperative bleeding. The appropriate treatment is to give 300 ml. of 3% saline I.V.

2. Diabetes insipidus may follow severe head injuries or intracranial surgery.

3. Fluid balance in the comatose or postoperative patient should be closely followed by daily weighing of the patient, careful recording of intake and output, and frequent determinations of blood electrolyte levels. Fortunately, in neurosurgical patients, nasogastric tube feeding is well tolerated and should be instituted as soon as possible to prevent the difficulties encountered with intravenous feedings.

CONGENITAL LESIONS
OF SURGICAL IMPORTANCE

A clear understanding of the embryology of the CNS is essential in evaluating congenital lesions involving the nervous system. The formation of the neural groove begins at the mid-embryo, and closure progresses both rostrally and caudally. As the tube is formed it separates from the ectoderm and comes to lie within the mesoderm. Since the nerve tissue is formed from the ectoderm and early comes to lie within the mesoderm, malformations of the nervous system are frequently accompanied by malformations of mesodermal tissue as well as the skin (ectodermal) and its append-ages.

CRANIUM BIFIDUM

Failure of the bones of the skull to unite at the midline is usually discovered at birth since the bones of the skull in the mid-line are easily palpable. The diagnosis should be substantiated by skull x-rays. Cranium bifidum must be differentiated from sepa-ration of normal sutures due to raised intracranial pressure. The defect is rarely of significant size unless there is associated pro-trusion of the dura, arachnoid, or brain (cranial meningocele or meningoencephalocele).

Treatment is not usually required. If the defect is extensive, cranioplasty is indicated to protect the underlying brain, utilizing bone, plastic, or tantalum mesh. This procedure should be de-ferred for about 4 years until the period of rapid growth of the skull bones has passed.

CRANIOSTENOSIS
(Craniosynostosis)

The cause of premature bony fusion of the cranial sutures is not known. Normally, the bones of the skull are separated at birth and become joined in a fibrous union at the suture lines after 3-6 months of age; bony union does not normally occur before the fifth or sixth decade.

Early recognition of premature fusion of the sutures is impor-tant because brain weight more than doubles in the first year of life. The growth of the skull is caused by the growth of the brain, and if the sutures fuse prematurely normal brain growth will be hampered. Surgical intervention is essential to permit normal mental develop-ment.

Clinical Findings.

The principal clinical feature of craniostenosis is deformity of the cranial contour; the type of deformity is dependent upon the suture or sutures involved, as follows (in order of frequency of occurrence): (1) sagittal suture - dolichocephaly or scaphocephaly; (2) coronal suture - brachycephaly; (3) all sutures - oxycephaly.

Treatment and Prognosis.

The only treatment is surgical. The operation consists of removing a linear strip of bone, 1-2 cm. wide, following the line of (and including) the involved suture. Polyethylene film or tubing is then sutured over the edge of the bone in an attempt to delay regrowth; the periosteum should be widely resected to further delay regrowth.

The outlook for normal mental development is good if the diagnosis is made during the first few months of life and surgery is done before the rapid increase in brain growth is impaired. The operation may have to be repeated, since regrowth sometimes occurs in spite of vigorous efforts to prevent it.

ENCEPHALOCELE

Grouped under this term are all lesions which cause protrusion of meninges (with or without neural tissue) and neural tissue from their normal intracranial location. About 75% of all encephaloceles are in the occipital area. Most protrude extracranially in the midline, but protrusion not uncommonly occurs in the nasopharynx, nasal cavity, or orbit.

The sac may be large, with a narrow stalk, but is more commonly sessile in shape. On examination it is difficult to determine if the mass contains neural structures or only fluid; transillumination with a bright light in a darkened room will show neural masses as shadows against the homogeneous red glow of the fluid.

The objective of surgery is to expose the neck of the mass and the cranial defect; to open the sac and replace neural contents within the cranial cavity (or amputate the neural tissue if it is excessive or abnormal); and to obtain a water-tight dural closure. Large bony defects can then be repaired with wire mesh, plastic, or bone, but this is rarely required.

DERMAL SINUS TRACTS

Dermal sinus tracts are due to incomplete embryonal separation of the neural tube from the overlying ectoderm; this creates a persistent connection between the skin and the CNS or its investing membranes or bone. Since closure and separation of the neural tube proceeds caudally and rostrally from about the midpoint of the embryo, persistent dermal sinus tracts occur most commonly at either extremity of the neural tube, i.e., the anterior or posterior neuropore. Midline cutaneous defects most commonly occur in the sacral area. Most sacral midline cutaneous defects are of no neurosurgical significance, as they extend no deeper than the sacral fascia (pilonidal cysts). If they extend into the subarachnoid space through a bony defect (spina bifida), the tract may serve as a pathway of infection; meningitis is a frequent complication.

The diagnosis is usually suggested by the accompanying cutaneous defect (dimple, port wine stain, hair tuft, etc.). There may be a history of recurrent bouts of meningitis.

Surgical excision should be done to prevent meningitis.

HYDROCEPHALUS

Hydrocephalus is enlargement of the CSF pathways as a result of an increase in the amount of fluid under elevated pressure. In infants this causes progressive enlargement of the head.

Hydrocephalus may be due to (1) a decrease in the normal rate of CSF absorption; (2) over-production of CSF (rare); (3) obstruction to the normal flow of fluid by a congenital lesion such as occlusion of the aqueduct of Sylvius, occlusion of the foramens of Luschka and Magendie, or obstruction of the basilar cisterns, as may occur in the Arnold-Chiari malformation. The absorptive bed over the surface of the hemisphere may function improperly, leading to a decrease in the normal rate of CSF absorption. Other causes include neoplasm and inflammation due to hemorrhage or infection.

Clinical Findings.

The diagnosis of hydrocephalus is made on the basis of abnormal and progressive enlargement of the head. If the diagnosis is in doubt, repeated measurements of the occipital bregmatic circumference should be compared with standard charts. The fontanel is usually wide, tense, and nonpulsating. The frontal bosses are prominent, and sclera may be visible above the iris in the natural position of gaze as the eyeballs are displaced downward by pressure on the thin orbital roofs. Symptoms of increased intracranial pressure are often present: irritability, vomiting, somnolence, etc. If the cortex is very thin, transillumination of the head with a bright light in a darkened room will permit passage of light through the head. Complete transillumination occurs in far-advanced hydrocephalus with only a shell of cortex remaining and in hydranencephaly (hydrocephalus with partial absence of brain). If complete transillumination occurs, attempts at surgical correction of hydrocephalus are not warranted. With reasonably thick cortical mantle remaining, no transillumination occurs.

Before a decision can be made regarding the proper therapy, the type of hydrocephalus must be established. In obstructive hydrocephalus there is no communication between the ventricular system and the absorptive bed. In communicating hydrocephalus there is free communication between the ventricles and the subarachnoid space in the spinal canal. To determine the type and, if possible, the cause of hydrocephalus, the following steps should be carried out with the patient in the lateral decubitus position: (1) Subdural taps should be done to make certain that enlargement of the head is not caused by subdural hematoma. A No. 20 short-beveled pediatric spinal needle is inserted into the subdural space through the lateral angle of the anterior fontanel or the coronal suture well away from the midline. (2) If no blood is recovered on subdural tap, the needle should be cautiously advanced until the ventricle is entered. The depth at which the ventricle is entered should be recorded, as this is a measurement of the thickness of the remaining cerebral mantle. A manometer is then fixed to the needle and the pressure recorded. One ml. of neutral PSP or indigo carmine is then injected into the ventricle. A lumbar puncture is immediately performed and the time required for the dye to appear in the lumbar space recorded. If no dye appears within 20 minutes, the hydrocephalus is said to be an obstructive type. Air

ventriculography is then carried out by replacing sufficient ventricular fluid with air in 10 ml. increments. This exchange is carried out until 30-50 ml. of fluid have been withdrawn. Skull x-rays are then taken with the head in various positions to demonstrate the entire ventricular system. The ventriculogram is done (1) to demonstrate the width of the cortex and (2) to rule out a neoplasm and, if possible, to establish more definitely the cause of the hydrocephalus. As a general rule, if the cortex is less than 1 cm. thick, the prognosis for normal development is poor and operative correction is probably not justified.

Treatment.

Occasional cases will arrest spontaneously. Repeated lumbar punctures (in communicating hydrocephalus) or ventricular taps (in obstructive hydrocephalus) can be used to tide the patient over until definitive management can be carried out. Numerous operative procedures have been devised, but none are uniformly successful. Removal of the choroid plexus is occasionally effective. Several shunting procedures are available to direct the CSF to an area where it may be excreted or absorbed. The choice of shunting procedure depends to some extent on the type of hydrocephalus: in obstructive hydrocephalus the fluid must be taken from the ventricular system, whereas in communicating hydrocephalus the fluid can be taken from the lumbar subarachnoid space. The fluid can then be shunted into either the peritoneal or the pleural cavity. A subarachnoid ureteral shunt to direct the fluid from the lumbar subarachnoid space into the ureter is effective in communicating hydrocephalus but entails the loss of a normal kidney and the difficulties that can arise because of the fluid and electrolyte loss since the CSF is thus excreted from the body.

At the present time the most promising procedure is to create a shunt directly from the ventricular system into the vena cava or the right atrium (ventriculo-caval or ventriculo-atrial shunt). A valve inserted in the tubing prevents reflux of blood into the ventricle. No fluid is lost from the body, and the absorption of the fluid is not dependent upon an absorptive bed, as is the case with pleural and peritoneal shunts. The disadvantages of this procedure are (1) that the long-term effects of maintaining the tubing in the vena cava or atrium are not yet known, and (2) the blockage of the system by clotting on the distal end which occasionally occurs.

Prognosis.

In hydrocephalus secondary to neoplasm or inflammation, the lesion causing the hydrocephalus determines the prognosis. The prognosis of congenital hydrocephalus has become more favorable with the development of the newer shunting procedures, but long-term follow-up will be required before positive statements can be made. Every effort should be made to perform surgery as early as possible, before the cortical mantle becomes so thin as to preclude normal mental development.

PORENCEPHALY

This is a circumscribed cavity in cerebral tissue which communicates with the ventricle. The most common clinical findings are seizures and, at times, enlargement of the head, which may be asymmetric, particularly as viewed by x-ray. Surgical treatment is indicated only when seizures cannot be controlled by anticonvulsant drug therapy.

ARNOLD-CHIARI MALFORMATION

This consists of (1) elongation and caudad projection of the cerebellar tonsils through the foramen magnum; (2) elongation (and kinking) of the medulla into the cervical canal, so that the fourth ventricle frequently opens into the cervical spinal subarachnoid space; and (3) downward displacement of the cervical spinal cord, so that the cervical nerve roots run upward to their foramens of exit instead of along their normal horizontal course. The etiology is not known, but in cases with spina bifida, meningocele, and meningomyelocele, fixation of the spinal cord at the level of the malformation probably prevents the normal cephalad migration of the cord and thus causes downward displacement of the structures in the posterior fossa.

Hydrocephalus is often present, as well as congenital anomalies of the cervical vertebrae, e.g., fusion of 1 or more of the cervical vertebrae, resulting in a short neck (brevis colli); lower cranial nerve palsies, cerebellar disturbances, and/or pyramidal and posterior column disturbances.

The hydrocephalus and other symptoms may be relieved by posterior fossa decompression with high cervical laminectomy. At that time the cerebellar tonsils can be lifted out of the cervical spinal cord to allow the normal flow of CSF around the basilar cisterns; occasionally it may be necessary to amputate the cerebellar tonsils. The adhesions between the cerebellum and the spinal cord can be lysed as an aid to reestablishment of normal CSF flow. If these measures are ineffective in relieving the hydrocephalus, a shunting procedure may be required.

PLATYBASIA
(Basilar Impression)

This is a developmental defect in which there is upward displacement of the cervical vertebral column into the base of the skull so that the odontoid process projects into the cranial cavity. Other bone anomalies are frequently associated, e.g., occipitalization of the atlas, misshapen foramen magnum, and fusion of the cervical vertebrae.

Symptoms rarely develop prior to adult life. Neurologic signs are those of compression of the cervical spinal cord, with weakness, ataxia, sensory loss, sphincter disturbances, etc. The diagnosis is established by x-rays of the skull and cervical vertebrae. Platybasia is said to be present when, on a good lateral view of the skull, the odontoid process is seen to project more than

5 mm. above a line drawn from the posterior rim of the foramen magnum to the posterior edge of the hard palate (Chamberlain's line).

Treatment is directed toward decompression of the area of the foramen magnum by suboccipital craniectomy and cervical laminectomy. Recovery of function is occasionally excellent, but in most cases arrest is all that can be accomplished.

SYRINGOMYELIA (AND SYRINGOBULBIA)

A syrinx is a central cavitation of the spinal cord and/or the brain stem. This is usually found in the cervical and upper thoracic cord, but it may occur also at various levels in the brain stem. Syringomyelia or syringobulbia is believed to be due either to congenital maldevelopment or neoplastic proliferation of the glial cells around the central canal of the spinal cord.

The classical clinical finding is the dissociated loss of pain and temperature sensation in a "jacket" distribution involving the upper extremities, shoulder girdles, and upper thorax, with relative preservation of touch perception. This is produced when the cavitation disrupts the decussating pain fibers in the commissures around the central canal. Associated findings are weakness of the muscles of the upper extremities, particularly the intrinsic muscles of the hands, with visible atrophy and fasciculations. Some degree of long tract involvement in the lower extremities is often present. X-rays of the cervical or thoracic spine may reveal erosion of the pedicles or increase in the interpedicular distances. Contrast myelography may demonstrate widening of the spinal canal at the involved segments, or the spinal cord may be so widened so as to produce a total block to the flow of the contrast medium.

Treatment is by laminectomy and decompression (with or without drainage of the cystic area by needle aspiration), myelotomy (incision into the cord through the posterior median fissure), or an attempt to establish permanent drainage of the cavity to slow the progression of the symptoms. X-ray therapy is advocated by some. The prognosis is guarded, although decompression and/or x-ray therapy may slow the progression of symptoms. The process seems at times to run its course with periods of relative quiescence.

SPINA BIFIDA OCCULTA

This is a defect in the laminar arch, usually asymptomatic, which most often occurs in the lumbosacral area. Associated anomalies in the skin overlying the bifid spine and malformations of the lower extremities (varus, valgus, or cavus deformities of the feet) are often present. Urinary incontinence, weakness, atrophy, sensory loss in the lower lumbar area and along the distribution of the sacral nerve roots, and other neurologic defects may be noted.

Contrast myelography may demonstrate an intraspinal mass, usually a lipoma.

Treatment is indicated only when there are associated neurologic findings, and consists of complete laminectomy with removal of any intraspinal mass (lipoma, dermoid, etc.). Care must be

taken to preserve nerve tissue. Electric stimulation is helpful in differentiating nerve filaments from fibrous strands. If a thickened filum terminale is discovered, this should be sectioned. The aim of surgery is to prevent further neurologic damage. Restoration of lost function is not likely to occur.

MENINGOCELE AND MYELOMENINGOCELE

A meningocele is a protrusion of meninges containing CSF through the defective neural arch. If neural elements are present, the lesion is then termed a myelomeningocele. All sizes and shapes of protrusions may occur, from a simple flat meningocele covered with a parchment membrane level with the skin to huge sessile masses with significant portions of neural tissue exposed on the surface (spinal rachischisis). They most commonly occur in the lumbosacral area, but may occur at any level and occasionally protrude anteriorly into the pelvic, abdominal, or thoracic cavities. It is often difficult or impossible to differentiate between simple meningocele and meningomyelocele, but transillumination of the mass with a bright light in a darkened room may reveal the shadows caused by neural elements. Neurologic deficits (sensory, motor, and sphincter disturbances) establish the lesion as a myelomeningocele.

The objective of surgery is cosmetic removal of the mass and prevention of further neurologic deficits. Surgery cannot restore function in nerve elements which never developed or which have been destroyed. The lesion should be removed in all cases if it is technically feasible and if the neurologic deficit does not preclude relatively normal development. Infants with spinal rachischisis and complete paraplegia should not be operated on until they are at least 6 months old; those who do not survive have overwhelming defects and associated anomalies which would have made them unacceptable candidates for surgery. A meningocele or meningomyelocele which has ruptured and is leaking CSF should be repaired on an emergency basis if the neurologic loss is not overwhelming. Otherwise in most instances operation can and should be delayed until there is sufficient skin covering the lesion to ensure a satisfactory skin closure. Progressive hydrocephalus should be surgically corrected before the repair of the meningocele, since increased CSF pressure threatens to break down the repair.

The prognosis depends upon the extent of the associated neurologic deficit and the severity of associated anomalies.

HEAD INJURIES

Injury to the head frequently results in brain injury, and the extent of brain damage determines the treatment and prognosis. The management of injuries to the scalp and skull is dictated by the type and degree of underlying brain damage and associated injuries in other parts of the body.

Emergency management is as follows:
1. Establish and maintain adequate respiratory exchange.
2. Control hemorrhage.
3. Treat shock.
4. Examine the patient quickly but thoroughly to ascertain the type and degree of all injuries.
5. Splint long bone fractures. **Note:** Do not reduce or apply casts; only splinting is warranted.
6. Evaluate the type and severity of the nervous system injury.
7. Do not move the patient for any reason (e.g., to obtain x-rays, for transportation to another hospital or to a private room) until the extent of all injuries is known and until the immediate threats to life (respiratory embarrassment, hemorrhage, etc.) have been controlled and the patient's condition stabilized.

Injury to the brain results from rapid deceleration, acceleration, and/or the shearing-rotational effects of a blow to the head. These mechanisms may produce (1) concussion, a temporary loss of consciousness with no permanent organic brain damage; (2) contusion, bruising of the brain; or (3) laceration, a frank disruption of brain substance. These 3 types of brain injury can occur singly, but are more commonly seen in varying combinations. Contusion may be local, causing focal signs and symptoms such as hemiparesis or aphasia; or generalized, with widespread damage to the brain. Increased vascular permeability in contusion produces cerebral "edema" or swelling. In severe head injuries decreased respiratory exchange leads to further anoxia and hypercapnia, resulting in cerebral vasodilatation, all of which contribute to further cerebral swelling.

Initial Evaluation of the Extent of Nervous System Injury.

Every case of head injury should be treated as potentially serious. As soon as practicable after the patient has been admitted to the hospital and his condition stabilized, a thorough neurologic examination should be performed. The following should always be noted and recorded.
A. Level of Responsiveness: Note response to spoken voice, shouted voice, and painful stimuli.
B. Size of pupils and response to light.
C. Ability to move the extremities voluntarily or in response to painful stimuli.
D. Degree and equality of the deep tendon reflexes.
E. Presence of superficial reflexes.
F. Presence of abnormal reflexes (e.g., Babinski response).
G. Direct inspection of the entire head for abrasions, swelling, laceration, etc.
H. Presence of bloody or CSF drainage from nose or ears.
I. Ophthalmoscopic examination, if possible. **Note:** Never artificially dilate the pupils in head injury.

The findings of this initial examination must be accurately recorded, as the indications for surgery depend to a great extent upon the observed variations from this initial baseline examination. The most important factor in management of severe head injuries is frequent observation, and the most sensitive sign of improvement or deterioration is change in the level of responsiveness. X-rays

of the skull should be taken in every case of significant injury to the head, but removal to the x-ray department should be deferred until the patient's condition is stabilized. X-rays of the cervical spine should be taken if there is evidence of injury to that area or if the patient is still unconscious on admission. Lumbar puncture should not be done unless specifically indicated.

GENERAL POSTOPERATIVE MANAGEMENT OF HEAD INJURIES

After definitive care of the more specific aspects of head injuries, one is frequently left with a comatose patient who has received a massive brain injury (contusions and/or lacerations). Hypoxia and hypercapnia must be prevented, since they cause intracranial vascular dilatation and add to cerebral swelling. Continuous intranasal oxygen is usually indicated, but frequent pharyngeal aspiration to keep the airway clear at all times and, if secretions accumulate rapidly, tracheostomy are the most effective measures to prevent hypoxia. Some neurosurgeons are now advocating prolonged hypothermia to a body temperature of approximately 31°C. as a means of preventing anoxic damage to the brain by reducing its oxygen requirement.

Intravenous urea, 150-300 ml. of 30% solution, can be used to reduce brain swelling, but should never be used until one is absolutely certain that there is no expanding intracranial clot; administration of urea will immediately expand a hematoma and produce disastrous results. Urea should therefore not be used until multiple bur hole exploration has ruled out hematoma.

For further details on the management of the comatose patient, see p. 14. Early application of rehabilitation technics by the physical therapy department may greatly shorten the period of rehabilitation.

SCALP INJURIES

The scalp is an extremely vascular structure, and injury may cause serious hemorrhage. Bleeding is usually best controlled with a simple pressure dressing of several 4 × 4 gauze sponges placed over the wound and held firmly in place by a circumferential dressing. Arterial bleeding can be controlled by firm finger pressure along the edges of the wound, or a hemostat can be attached to the galea and allowed to hang down over the skin edge in such a way as to pull the galea firmly against the skin and compress the bleeding vessels. All wounds of the scalp should be closed as soon as possible unless they overly a depressed fracture or penetrating wound of the skull. If either of these is present, the patient should be taken to the operating room and the skin wound treated in conjunction with the definitive treatment of the fracture.

Scalp Lacerations.

Always shave at least 1 inch beyond all edges of the wound. Wash the area around the wound thoroughly with surgical soap, taking care to wash away from the wound. (If the laceration is large it is best to place a 4 × 4 gauze sponge in the wound to prevent debris

from entering.) Cleanse the wound thoroughly with repeated irriga-
tions of warm normal saline or Ringer's solution. Infiltrate the
circumference of the wound with local anesthetic about 1 inch away
from the wound edges. Remove foreign bodies which may lead to
infection or leave unsightly tattoos. Minimal debridement should
include all devitalized tissue, taking care not to cut away so much
tissue that closure will be difficult or impossible.

The wound can be closed with multiple interrupted vertical
mattress sutures of stainless steel wire or with a double layer of
interrupted sutures of the galea and skin. Technic should be me-
ticulous.

Avulsions of the Scalp.

Avulsions of the scalp usually include all layers down to the
periosteum. These injuries should be treated only by experienced
personnel with complete operating room facilities. The surrounding
area should be shaved and thoroughly irrigated. If the avulsion is
small, the ragged edges can be sparingly trimmed; closure can
often be accomplished by tripod extension or modified Z-plasties.
If the denuded area is large the surrounding area should be shaved;
the wound is then repeatedly and thoroughly irrigated and covered
with a single layer of fine mesh (burn) gauze. A large dressing of
"fluffies" or mechanic's waste is placed over this and a firm cir-
cumferential dressing applied to exert even pressure over the area.
Delayed closure via a plastic procedure will then be required 8-10
days later. If the bone is completely denuded of all soft tissue, a
small split-thickness graft can be applied as a temporary measure
and covered with lightly impregnated fine mesh gauze to protect the
bone until delayed closure can be safely carried out.

SKULL FRACTURES

Skull fracture per se is not a more serious injury than fracture
of any other bone; it is the injury to the brain that determines the
prognosis in head injuries. Death due to severe brain injury may
follow head injury in which there is no evidence of skull fracture;
and patients with extensive "egg-shell" fractures of the skull may
have no neurologic deficit or other permanent ill effects.

Fractures may be classified according to (1) whether the skin
overlying the fracture is intact (simple) or broken (compound); (2)
whether there is a single fracture line (linear), several fractures
radiating from a central point (stellate), or fragmentation of bone
(comminuted); and (3) whether the edges of the fracture line have
been driven below the level of the surrounding bone (depressed) or
not (nondepressed).

Simple Skull Fractures (Linear, Stellate, or Comminuted Non-
depressed).

These fractures are of clinical importance if they have crossed
major vascular channels in the skull such as the groove of the mid-
dle meningeal artery or the major dural venous sinuses. If these
vessels are torn, epidural or subdural hematomas may form; and
all patients with these types of fractures should be kept under close
observation until it can be ascertained that no such bleeding is
occurring. A fracture which extends into the accessory nasal

sinuses or the mastoid air cells is considered to be compound, since it is in communication with the external surface of the body.

Depressed Skull Fractures.

Depressed stellate or comminuted fractures require surgical elevation, but if there are no untoward neurologic signs and the fracture is closed, surgery can be delayed until a convenient time.

Compound Skull Fractures.

As soon as his general condition warrants, the patient with a depressed compound skull fracture should go to surgery where complete neurosurgical facilities and a trained neurosurgeon are available. Emergency treatment consists merely of applying a sterile compression dressing. The wound should not be closed, and no attempt should be made to remove any foreign body protruding from the wound until the patient is in the operating room and all preparations have been made for craniotomy, as catastrophic hemorrhage may occur when the tamponading effect of the foreign body is no longer present.

Linear or stellate, nondepressed compound fractures can be treated by simple closure of the skin wound after thorough cleansing. Compound fractures with severe comminution of underlying bone should be treated in the operating room, where proper debridement can be carried out. Mature judgment is required in deciding whether small pieces of bone completely denuded of blood supply should be removed before the wound is closed. If possible, the dura should be inspected to make certain that no laceration has been overlooked. Basal skull fractures may cause rhinorrhea or otorrhea if the dura and arachnoid are torn, and may also cause bleeding from the nose or ears if mucous membranes or skin are torn. When such bleeding occurs it is often difficult to tell if blood is admixed with CSF. A presumptive diagnosis of CSF leakage can be made by placing a drop of the bloody discharge on a cleansing tissue; if CSF is present there will be a spreading yellowish-orange ring around the central red stain of blood. A patient with otorrhea or rhinorrhea should be kept in bed for at least 10 days with his head elevated about 35° and strongly cautioned not to blow his nose for fear of forcing contaminated material from the nose or ear into the cranial cavity. Prophylactic antibiotics may be used. In about 80% of cases, these CSF leaks will close spontaneously within 8-10 days. If CSF drainage persists, the point of leakage should be ascertained and repaired via craniotomy.

TRAUMA TO THE MENINGES

Head injury with or without skull fracture may cause tears in the major or minor vascular channels coursing through the meninges and lead to serious intracranial hemorrhage. Tears in the dura should always be repaired to afford water-tight closure (to lessen the chance of infection or epileptogenic scar formation).

EPIDURAL HEMATOMA

Hemorrhage between the inner table of the skull and the dura mater most commonly arises from a tear in the middle meningeal artery produced by a linear fracture in the temporal region when the fracture line crosses the arterial groove. Since the bleeding is under arterial pressure, it gradually strips the dura from the under-surface of the bone; this produces more bleeding as the small bridging veins from dura to bone are torn. The hematoma thus in-creases in size rapidly and severely compresses the cerebral cor-tex, producing contralateral hemiparesis or hemiplegia. As the hemisphere is further compressed, the medial portion of the tem-poral lobe (hippocampus) is forced through the incisura tentorii (compressing the third cranial nerve and thus producing dilatation of the pupil on the same side) and the brain stem is shifted toward the opposite side of the tentorial notch. If the brain stem compression reaches significant degree, venous hemorrhages into the stem will lead to irreparable neurologic deficits or death.

In rare cases epidural hematoma may arise from venous chan-nels in the bone at the point of fracture or from lacerated major dural venous sinuses. Since venous pressure is too low to dissect the dura from the bone, venous hematomas usually form only when a depressed skull fracture has stripped the dura from the bone and left a space in which the hematoma can form.

The classic case presents a history of a blow to the head (which may or may not be of sufficient force to bruise the overlying skin), leading to unconsciousness for a brief interval. As the patient regains consciousness he may have no untoward neurologic symptoms and be perfectly clear mentally; this is the so-called lucid interval. In a matter of minutes or hours, as the rapidly forming hematoma enlarges sufficiently to severely compress the cerebral hemisphere, there is a gradual deterioration of conscious level progressing to coma and death if the hematoma is not imme-diately evacuated. As the conscious level deteriorates the pupil on the side of the lesion dilates and a contralateral hemiplegia occurs.

The mortality rate in epidural hematoma is high despite the fact that this is potentially a completely curable lesion. The prob-lem is that these patients are either not seen by a physician (since head injury is felt to be too mild) or, more commonly, because they are seen during the lucid interval and discharged to bed rest at home. The patient is then assumed to be asleep when actually the hematoma has increased in size, producing coma instead of sleep; not uncommonly this type of patient is found dead in bed the next morning. Any patient with a history of a blow on the head leading to unconsciousness, even for a brief interval, should therefore have a thorough neurologic examination and skull x-rays. If x-rays show a fracture, he should be hospitalized and his conscious level checked at least every hour. Any impairment of the sensorium is an indication for immediate bur hole exploration in the operating room. This is a true emergency, for if surgery is delayed until brain stem hemorrhages occur the patient is likely to remain decerebrate even though the clot is surgically evacuated.

In those cases with a history of a blow to the head followed by unconsciousness, even though no fracture is demonstrated, a re-liable relative should be instructed to awaken the patient at least hourly to make certain he is arousable and not comatose.

SUBDURAL HEMATOMA

Subdural hematomas occur most commonly (1) when the veins running from the cortex through the subdural space to the superior sagittal sinus near the midline are torn or (2) when an intracerebral hematoma communicates with the subdural space. The bleeding then occurs between the arachnoid and the dura into the subdural space. Since the arachnoid is only loosely attached to the dura, these hematomas can attain tremendous size even though the bleeding is of venous (low-pressure) origin.

Acute Subdural Hematomas.

Acute subdural hematomas occur only in severe head injuries, probably as a result of frank lacerations of the pia and arachnoid of the cortex and subsequent bleeding into the subdural space. They are usually thin, as the overwhelming injury to the brain often produces cerebral swelling, frequently with great increase in pressure. This makes it impossible for a hematoma of venous origin to attain significant size. These lesions are usually discovered during bur hole exploratory surgery on severely injured patients. Evacuation rarely produces significant change in the patient's condition, as the major neurologic deficit is produced by the widespread cerebral contusion and/or laceration.

Subacute Subdural Hematomas.

These are also due to brain contusion and laceration. Because the brain injury is not overwhelming and brain swelling not great, the hematoma may become large enough to produce symptoms. Progressive hemiparesis, obtundation, aphasia, etc. often appear 3-10 days after the injury. Evacuation of the clots may improve the patient's condition, and the degree of ultimate recovery depends upon the extent of brain damage produced at the moment of injury.

Chronic Subdural Hematomas.

Chronic subdural hematomas are most common in infants and in adults past middle age. They are commonly produced by tearing of the veins that bridge the subdural space as they pass from the convexity of the cortex into the superior sagittal sinus. The hematoma becomes encased in a fibrous membrane, usually liquifies, and gradually enlarges. A history of head injury may be lacking, as the blow is often quite minimal. The history is usually one of progressive mental or personality changes with or without focal symptoms (progressive hemiplegia, aphasia, etc.). Papilledema may be present. These findings often suggest a diagnosis of brain tumor, and the hematoma may be discovered by angiography or bur hole placement preparatory to ventriculography. Subdural hematomas can mimic almost any disease affecting the brain and its coverings.

Treatment consists of drainage of the clot or fluid contained in the subdural space. Many feel that evacuation through multiple bur holes is sufficient; others advocate evacuation through a craniotomy so that the subdural membranes (fibrous capsule) can be widely resected to offer a better prognosis for return of normal mental function and to discourage re-formation of the hematoma.

Subdural Hygromas.

These are collections of clear or yellow-stained fluid in the subdural space. Their mechanism of production has not been completely explained, but most neurosurgeons agree that they form through a tear in the arachnoid which allows CSF to escape into the subdural space and to be held there by a postulated ball valve action of the tear.

Hygromas produce symptoms of subdural hematoma.

Treatment consists of draining the subdural space through multiple bur holes.

Subarachnoid Hemorrhage.

Traumatic laceration of vessels in the subarachnoid space is the most common cause of subarachnoid hemorrhage. The diagnosis can be made by collection of uniformly bloody CSF on lumbar puncture, or on the basis of nuchal rigidity, positive straight leg raising tests, and a positive Brudzinski response. No surgical treatment is indicated.

BRAIN INJURY

Direct injuries to the brain, as contusions or lacerations, are not amenable to surgical treatment. Some neurosurgeons feel that in profound injuries, where experience has shown that the temporal and frontal lobe tips are the most seriously injured portions, resection of these severely contused portions of cerebrum may be indicated. Intracerebral hematoma may occur as a result of trauma, usually in association with lacerations; if they are of significant size, surgical evacuation is indicated.

INJURIES TO THE SPINE*

Fractures and fracture-dislocations of the vertebrae are usually the result of acute hyperextension and hyperflexion of the spine. Penetrating injuries may occur as a result of gunshot or stab wounds, etc. Concussion of the spinal cord produces temporary physiological interruption of function without anatomically demonstrable changes. Contusion of the cord usually occurs at the site of a fracture-dislocation or penetrating wound. Laceration, disruption, or dissolution of part or all of the spinal cord is usually the result of severe fracture-dislocations or penetrating wounds.

Clinical Findings.

The neurologic loss in trauma to the spinal column may be partial or complete and transient or permanent. The usual clinical findings are as follows: (1) Immediate flaccid paralysis of the extremities and complete areflexia below the level of the injury due to "spinal shock." Spinal shock may persist for hours, days, or months, depending upon the severity of the injury and the presence

*Only those spine injuries which are accompanied by neurologic manifestations will be discussed here. See also Chapter 19, Traumatic Orthopedic Surgery.

of anemia, hypoproteinemia, urinary tract infection, etc. (2) Complete or partial loss of any or all modalities of sensation below the level of the lesion. (3) Urinary retention. (4) Paralytic ileus with abdominal distention. (5) In cervical lesions below C4, there is frequently respiratory difficulty since in this situation the intercostal musculature is paralyzed and breathing is purely diaphragmatic. (6) Loss of sweating below the level of the lesion. (7) Point tenderness over the fracture site, with or without gibbus or crepitus. (8) X-rays may be negative or may demonstrate simple fracture or complete fracture dislocation with marked comminution.

Diagnosis is usually not difficult, but in severe brain injuries with loss of consciousness areflexia is frequently present initially and spinal cord injury may be overlooked. It is imperative in all severe head injuries that x-rays be taken of the cervical spine to be certain that cervical fracture-dislocation is not present.

Treatment.

A thorough neurologic examination must be performed as soon as possible after injury to establish the level and degree of functional loss. Spinal cord injury must be assumed until there is definite proof that none has occurred. Do not move the patient until the full extent of the injury is known, since the spinal cord may be irrevocably damaged by careless handling.

Obtain a detailed description of the accident, as management may depend upon whether the injury was caused by hyperflexion, hyperextension, or a direct blow. Examine for associated injuries.

If cervical fracture is suspected, place sandbags at both sides of the head to prevent motion and apply halter traction with 6-10 lb. of weight as emergency splinting measures. At least 4 and preferably 6 persons are required to lift the patient onto a Stryker or Foster turning frame, taking care to allow no distortion of the spine.

Ask the patient to void; if he is unable to do so, catheterize with a No. 14 or No. 16 Foley catheter and leave it in place.

As soon as the patient's general condition warrants, obtain adequate x-rays of the spine. The physician should always accompany the patient to the x-ray department and remain with him until satisfactory anteroposterior and lateral views have been obtained. The patient should not be turned for the purpose of x-ray, as adequate projections can be obtained with the cross-table technic.

If a cervical fracture or fracture-dislocation is found on x-ray, skeletal tong traction should be immediately instituted. This can be carried out in the x-ray room or, preferably, in an operating room which has an x-ray unit.

Some neurosurgeons feel that laminectomy is almost never indicated; others feel it is always indicated. However, the weight of authoritative opinion is that laminectomy should be done in any of the following circumstances: (1) if there is progressive neurologic loss, (2) if total block of the vertebral canal is shown by a positive Queckenstedt test, (3) if x-rays show bone fragments or foreign bodies depressed into the spinal canal, or (4) if there is a direct penetrating missile wound of the spinal column. If laminectomy is decided upon, it should be carried out as soon as the patient's condition is stable and immediately life-threatening associated injuries have been dealt with. If the injury is cervical, skeletal tong traction should be instituted immediately and maintained during surgery.

PROTRUDED (HERNIATED)
INTERVERTEBRAL DISKS

The protrusion may consist of a posterior or lateral bulge of the weakened but intact annulus fibrosus produced by a partially dislocated nucleus pulposus, or part of the entire nucleus may extrude into the spinal canal through a rupture in the annulus.

PROTRUSION OF LUMBAR INTERVERTEBRAL DISKS

Clinical Findings.
  A. Symptoms and Signs: All but 5-10% of protrusions occur at the last 2 lumbar interspaces. There is usually a history of progressively more prolonged episodes of back pain with or without leg pain. The pain characteristically spreads to the gluteal region and then down the posterior or posterolateral aspect of the thigh and calf; is aggravated by coughing, sneezing, or straining; and may be relieved by rest. The patient will limp to favor the painful leg, and will tend to carry himself with a "list" either away from or toward the side of the protrusion. Other significant symptoms and signs include spasm of the paravertebral muscles; flattening of the lumbar spine with loss of the normal lordosis; limitation of back motions by pain, particularly on forward flexion; and tenderness to deep pressure over the interspace involved.
  B. Neurologic Examination:
    1. Straight leg raising produces leg pain and frequently back pain which can be accentuated by dorsiflexion of the foot (further stretching of the sciatic nerve and roots).
    2. Reflexes - The ankle reflex is diminished or absent (more common with protrusion at the L5-S1 interspace impinging on the first sacral nerve root). The knee reflex may be diminished with herniations at the L3-L4 interspace.
    3. Motor evaluation - Weakness of dorsiflexion, plantar flexion, and inversion or eversion of the foot may be found with herniations at the lower 2 lumbar interspaces. Weakness of the quadriceps may be present with L3-L4 lesions.
    4. Sensory evaluation - Sensory status varies from no loss to complete analgesia in the distribution of the root involved.
  C. X-ray Findings: Plain films may be normal or may show narrowing of the involved interspace, arthritic "lipping" of the adjacent vertebral bodies, and/or arthritic changes around the zygoapophyseal joints.
  D. Special Examinations:
    1. Myelography is the most commonly used and most valuable test. Iophendylate, N.N.D. (Pantopaque®), is the contrast medium most often used. Three to 21 ml. of medium are instilled into the lumbar thecal sac and the dye observed fluoroscopically as it flows over the suspected interspaces. Permanent films record the extent and level of the lesion.
    2. Electromyography can be used to identify the involved nerve root by demonstrating denervation fibrillation potentials in the muscles supplied by the root.

3. Diskograms are rarely employed. Diatrizoate (Hypaque®)
   is injected directly into the disk. A normal disk will accept
   0.5 ml. or less; pathologic disks may accept 2 or 3 ml., and
   injection frequently reproduces the patient's clinical pain.
   X-rays confirm disruption of the disk by demonstrating
   diffusion of opaque material.

Differential Diagnosis.
   Protrusion of lumbar intervertebral disks must be differentiated
from tumor, subluxation of facets, and rheumatoid spondylitis.

Treatment.
   A. Conservative Measures: Many cases can be tided over an acute
      episode by conservative measures. Supportive treatment in-
      cludes analgesics for pain and strict bed rest with boards be-
      tween springs and mattress to prevent sagging. Bilateral leg
      traction or pelvic traction may help to reduce muscle spasm.
      The patient is often more comfortable in the modified Fowler's
      or contour position. After the acute pain subsides, a progres-
      sive resistance physical therapy program should be instituted
      to strengthen the back, abdominal, and leg muscles and to in-
      struct the patient in the proper way of using his back muscles
      to prevent straining injuries. Back support with a corset brace
      or flexion body cast may be useful when back pain is severe
      and chronic.
   B. Surgical Measures: Surgery is indicated when complaints are
      too severe for conservative treatment or when there are pro-
      gressive signs of neurologic involvement. If the patient does
      not respond favorably to a trial of conservative treatment,
      laminectomy and simple excision of protruding nucleus pulposus
      is sufficient in most cases.

PROTRUSION OF CERVICAL INTERVERTEBRAL DISKS

Clinical Findings.
   A. Lateral Protrusions: Patients with lateral cervical protrusions
      give a history of frequent bouts of pain involving the neck,
      shoulder, and scapular region (ultimately accompanied by
      lancinating root pain into the arm or hand) which is accentuated
      by straining. Paresthesias into the fingers are common: into
      the first and second digits if the root of C6 is involved; the
      third digit if the root of C7 is involved; and the fourth and fifth
      digits if C8 is involved. Examination reveals restricted neck
      motions, absense of cervical lordosis, spasm of the neck
      muscles, hypesthesia in root distribution to the arm and hand,
      and weakness in the muscle supplied by the involved root.
      Arm reflexes are often decreased: diminished biceps reflex if
      C6 is involved; diminished biceps and triceps reflexes if C7 is
      involved; and diminished triceps reflex if C8 is involved.
         X-rays may be normal if the protrusion is recent, or may
      show a narrowed interspace and/or hypertrophic lipping and
      narrowing of the root foramens. Iophendylate (Pantopaque®)
      myelography (see p. 510) reveals a defect of the root sleeve.
   B. Midline Protrusions: With midline protrusions there may be

no history of root pain; a common presenting complaint is spastic paraparesis with or without urinary hesitancy or incontinence. Signs of pyramidal tract involvement are apparent in the lower extremities; if the cervical roots are involved there will be signs of muscle weakness or atrophy and reflex and/or sensory changes in the upper extremities. Less commonly, there is impairment of ability to perceive pinprick up to the high thoracic level due to spinal cord compression. X-rays reveal marked interspace narrowing, often at several levels, with extensive bony proliferation at the posterior edges of the vertebrae. Myelography shows midline bar deformities.

Treatment.
A. Conservative Measures: Bed rest with continuous halter traction initially; as symptoms subside, a felt or metal neck brace may be helpful.
B. Surgical Measures: In lateral protrusions simple removal of the fragment through the posterior hemilaminectomy approach gives good results. In midline protrusions the operation of choice in recent years has been laminectomy over the involved area with sectioning of the dentate ligaments on both sides to allow the spinal cord to be displaced posteriorly and thereby relieve the anterior compression on the cord. Anterior diskectomy and intervertebral fusion (through the paratracheal approach) has produced excellent results in both lateral and midline disk protrusions, but long-term follow-up studies on these patients are not yet available. In midline bar protrusions surgery usually prevents progression of neurologic loss and may partially restore lost function.

PERIPHERAL NERVE INJURIES

Injuries to the peripheral nerves may be caused by lacerating injuries, fractures of the long bones, crushing injuries of the extremities, or penetrating or perforating wounds. These injuries may cause partial or complete anatomic separation of the nerve, destruction of the nerve with the nerve sheath remaining in continuity (as in crush injuries), or temporary impairment of function without actual destruction of nerve tissue (as in concussion injuries).

Clinical Findings.
' Trauma to the peripheral nerves causes complete or partial loss of sensory and/or motor function in the structures innervated by the nerve involved. The diagnosis is made by individual testing of the muscles in the involved extremity to determine which are paretic and to what extent. The area of sensory loss should be outlined on the skin and a photograph or drawing entered in the record. There may be extremely painful dysesthesias referred to hypalgesic areas. After the acute phase of the injury has passed, percussion over the stump or suture site evokes a tingling sensation which is projected into the anesthetic area in the distribution appropriate to the nerve involved (Tinel's sign). In old nerve injuries the nails and skin may show severe trophic changes, and painless ulcers may be present as a result of injuries or burns to anesthetic skin.

After 21 days (the time required for complete degeneration of nerve distal to the point of injury), electromyography shows denervation fibrillation potentials, increased chronaxie, failure to react to faradic stimulation, and absence of voluntary contraction.

Treatment.

A. Initial Treatment of Acute Injuries: If the patient is seen within 6-8 hours after injury and there is little surrounding soft tissue injury and minimal contamination, primary end-to-end anastomosis can be carried out in the operating room (never in the emergency room). If the injury is of the crushing type or has resulted in a jagged laceration with a great deal of soft tissue injury, no attempt should be made to carry out primary anastomosis. In these cases the nerve ends (distal and proximal) should be tagged with wire sutures for later identification and the wound treated appropriately; delayed anastomosis can then be carried out 18-21 days later, at which time degeneration of nerve distal to the wound will be complete and the Schwann cell tubules will be ready to accept outgrowing neurofibrils. At this time it will be possible to determine how much of the nerve ends are nonviable, resect back to healthy nerve, and, if necessary, mobilize the nerve above and below so that end-to-end anastomosis can be carried out.

B. Old Nerve Injuries: If the initial injury resulted in complete loss of function and primary nerve repair was not attempted, anastomosis will usually give good results as long as 1-2 years after the injury. In incomplete nerve injuries seen weeks or months after injury, mature judgment is required to decide which of the following surgical approaches is indicated: (1) extrinsic neurolysis (lysis of scar external to the nerve) and/or intrinsic neurolysis (lysis of scar within the nerve sheath); or (2) resection of the neuroma followed by neurorrhaphy.

CENTRAL NERVOUS SYSTEM NEOPLASMS

INTRACRANIAL NEOPLASMS

Brain tumors occur in all age groups with approximately equal distribution up to the age of 70 years. Two-thirds of all brain tumors in children occur in the subtentorial space (posterior fossa), whereas 80% of brain tumors in adults occur in the supratentorial space. Certain brain tumors are found almost exclusively in childhood (medulloblastoma); others are found almost exclusively in adults (glioblastoma multiforme). Brain tumors almost never metastasize outside the CNS. Brain tumors are found in 1% of hospital admissions and perhaps as many as 2% of autopsies.

Classification and Approximate Frequency.

Gliomas, 45%; meningiomas, 15%; pituitary tumors, 12%; tumors of nerve sheaths, 8%; blood vessel tumors, 5%; congenital tumors, 5%; and metastatic tumors, 2-5%. Miscellaneous tumors account for the remainder.

Clinical Manifestations of Brain Tumors.

The diagnosis of brain tumor is made on the basis of the history, neurologic examination, and appropriate laboratory tests, and is confirmed by special diagnostic tests. In any suspected case of brain tumor a careful ophthalmoscopic examination should be made. If papilledema is present, spinal puncture should not be done. If papilledema is not present and a brain tumor is suspected, a carefully performed spinal puncture can be quite helpful, since by carefully measuring the pressure one can determine if increased pressure is present which has as yet not produced papilledema. One or a combination of the special diagnostic tests discussed below are usually carried out to confirm a strong suspicion of brain tumor, to localize the lesion as accurately as possible, and to gain as much information as possible preoperatively concerning the character of the tumor.

A. Symptoms and Signs: These depend upon the location and rapidity of growth of the tumor and the age of the patient. Symptoms and signs are produced by the local mass effect of the tumor or by secondary effects such as edema and obstruction of the flow of CSF.

1. General or secondary effect - The increased intracranial pressure produced by any space-occupying intracranial mass leads to the classic triad of (1) headache, commonly located in the vertex, worse in the morning after prolonged recumbency, and frequently relieved by standing up; (2) nausea and vomiting; and (3) papilledema. Increased intracranial pressure may also cause personality changes, convulsions, cranial nerve palsies, and even homonymous hemianopsia as the posterior cerebral artery passing over the edge of the tentorium is compressed by the tense brain. These signs and symptoms, then, are not necessarily of localizing or even lateralizing value since they can be produced by pressure alone. The focal signs of the brain tumor depend upon its location (e.g., temporal lobe, cerebellum, third ventricle) and on whether it is situated on the surface or in the deeper tissues.

The most common presenting complaint is also dependent upon the location of the tumor, its cell type, rapidity of growth, etc. Whether the presenting complaint is headache, convulsions, hemiparesis, or visual or gait disturbances, it is imperative to determine if there is a history of progression - no matter how rapid or slow - as it is progression of symptomatology which justifies the strong suspicion of brain tumor and the confirmatory studies outlined below.

2. Focal effects - In diagnosing brain tumors, one asks first where the lesion is and second what type of lesion it is. The clinical manifestations of tumors in various locations in the brain are discussed below as a guide to determining where the lesion is; the remainder of this section will be devoted to brief discussions of individual types of tumors as a guide to determining what type of lesion it may be.

a. Frontal lobe - Involvement of the frontal lobes usually produces personality changes (inappropriate behavior, loss of social inhibitions) and mental changes. These findings are easily confused with those produced by

increased pressure, but if increased pressure is present there will also be headache, nausea and vomiting, and papilledema. If the posterior portions of the frontal lobe are involved, the patient may show forced grasping and, if the motor areas of the cortex or subcortical areas are involved, varying degrees of hemiparesis on the contralateral side, increased deep tendon reflexes, and a positive Babinski reflex; if the tumor is in the dominant frontal lobe, expressive aphasia may be manifest. If convulsions occur they are frequently of the adversive type (head and eyes turned toward the side opposite the lesion). Tumors arising anteriorly beneath the frontal lobes may cause anosmia.

b. Parietal lobe - Involvement of the parietal lobe tends to produce contralateral paralysis accompanied by defects in the appreciation of the weight, texture, size, and shape of objects. Ability to perceive and localize pinprick on the side of the body opposite the lesion may be impaired (astereognosis). Parasagittal tumors (involving the paracentral area) may cause spastic paralysis of the contralateral leg or, as the tumor enlarges, paraplegia and urinary incontinence. Tumors low in the parietal area may produce visual field defects. If the dominant hemisphere is involved, global aphasia will be present.

c. Occipital lobe - Occipital lobe tumors are relatively uncommon. Visual field defects are the major symptom, and tend to be more congruous than visual field defects produced by lesions in the temporal lobe. (Complete homonymous hemianopsia has no localizing value since it can be produced by a lesion anywhere from the chiasm to the occipital lobe.) Isolated visual hallucinations may occur, or they may appear as part of a generalized seizure.

d. Temporal lobe - "Temporal lobe seizures" are a common manifestation of temporal lobe tumors. There may be classical uncinate fits accompanied by unpleasant olfactory sensations, often with lip smacking and loss of contact with surroundings. A generalized seizure may follow. The seizure may take the form of momentary episodes of staring of which the patient is not aware, a feeling of unreality, and extreme familiarity (déjà vu) or unfamiliarity with surroundings at the time of the attack. Distorted perceptions of sounds, objects, sizes, or shapes may be noted. Visual field defects are common and tend to be incongruous. If the dominant temporal lobe is involved, receptive aphasia or auditory agnosia may occur.

e. Posterior fossa - Tumors in the posterior fossa produce early symptoms by involvement of the cerebellum, brain stem, and cranial nerves, and, as a rule, by obstruction of the flow of CSF.

   (1) Cerebellum - A tumor arising in a hemisphere of the cerebellum causes ataxia of gait and incoordination of the ipsilateral arm and leg. Nystagmus is common. The principal manifestation of a tumor involving

primarily the midline of the cerebellum is often ataxia of the trunk, with unsteadiness and falling even while sitting; incoordination of the arms is less frequent. Cranial nerve palsies, particularly of those nerves innervating the extraocular muscles, are prone to occur.

(2) Brain stem (intramedullary) tumors produce increased intracranial pressure late; the most common symptoms and signs are multiple cranial nerve palsies, nystagmus, and incoordination and paresis of extremities.

(3) Cerebellopontine angle tumors (presenting in the angle formed by the petrous ridge, tentorium, cerebellum, and brain stem) - The vast majority arise from the vestibular portion of the eighth cranial nerve (e.g., "perineural fibroblastoma," neurinomas, neurilemmomas). The classical symptomatology is tinnitus on the involved side followed by nerve deafness. Involvement of the seventh cranial nerve produces facial palsy; involvement of the fifth cranial nerve may give rise to numbness or paresthesias in the face and loss of the corneal reflex. Involvement of the cerebellum causes ataxia, dysmetria, and nystagmus.

(4) The midbrain is usually involved secondarily by tumors arising from nearby structures (e.g., the pineal gland), but may be involved directly. Tumors in the pineal region frequently produce direct pressure on the roof structures of the midbrain, difficulty in upward gaze (Parinaud's syndrome), and impairment of the pupillary light reflex; involvement of the red nucleus area produces ataxia, incoordination, and intention tremor.

f. Sellar region - Tumors arising in the pituitary gland may produce hyperfunction of portions of the gland (acromegaly) or, more commonly, hypofunction, leading to loss of libido, pubic and axillary hair, etc. Appropriate laboratory tests confirm the hypopituitarism. As sellar tumors enlarge they encroach on the optic chiasm and produce a characteristic bitemporal hemianopsia.

B. Special Examinations:

1. Pneumoencephalography - With the patient in the sitting position and usually under local anesthesia, a spinal puncture is carried out and a measured amount of air is introduced (and a comparable amount of fluid withdrawn) to adequately fill the ventricular system and subarachnoid pathways to give good visualization of the intracranial structures. Multiple x-rays are taken of the skull in varying positions. This test is never used if intracranial pressure is known to be increased.

2. Ventriculography - Utilizing local anesthesia, bur holes are placed in symmetric positions, 4 cm. to each side of the midline in the posterior parietal region, and special needles introduced into the atrial region of the lateral ventricles. Air is then exchanged for the fluid and multiple x-rays are taken with the skull in various positions. This test is used when there is increased intracranial pressure.

3. Carotid and/or vertebral arteriography - The appropriate artery is injected with a contrast medium (50% Hypaque®)

while serial x-rays are rapidly taken. This test is used to localize the tumor, either by indicating a tumor "blush," or by inference, indicating which arteries or veins are shifted out of their normal position.
4. Electroencephalography - This is helpful in the recognition of certain brain tumors involving the cerebrum since it may indicate a focal pathologic process. The localization thus arrived at is never accurate enough for purposes of surgical treatment.

Treatment of Brain Tumors.

In general, the treatment of all brain tumors is surgical exploration and excision through a craniotomy or craniectomy centered as accurately as possible over the tumor. The tumor should be completely removed if this can be done without producing a serious neurologic deficit. If total removal is not possible, subtotal removal should be as radical as is feasible for the purpose of decompression. If the location of the tumor precludes direct surgical attack (e. g., third ventricle or pontine) and it blocks the flow of CSF, shunting of the CSF from the lateral ventricles to the cisterna magna (Torkildsen procedure) should be carried out. Postoperative x-ray radiation may be of considerable value in some tumors and of no value in others.

INTRACRANIAL GLIOMAS

All tumors which arise from the interstitial cells of the CNS are included in this category.

Glioblastoma Multiforme.

Glioblastoma multiforme (about 25% of gliomas), the most malignant of all primary tumors of the brain, arises in the white matter, grows rapidly, and has a strong tendency to cross to the opposite hemisphere. These tumors are highly cellular and pleomorphic, with many mitoses. The most common sites of occurrence are the frontal, parietal, and temporal lobes, and the highest incidence is in the age group from 40 to 60.

Glioblastoma multiforme can rarely (if ever) be completely removed, and is resistant to x-ray therapy. Life expectancy is usually less than 1 year after diagnosis.

Astrocytoma.

Astrocytomas make up approximately 35% of all gliomas. They are composed of adult astrocytes, are relatively slow-growing, and frequently blend diffusely with the surrounding brain. In children they occur predominantly in the cerebellar hemisphere and are often cystic. In adults, astrocytoma occurs most often in the frontal, parietal, and temporal lobes.

In adults these tumors are often impossible to remove completely, as they extend into vital deep structures. Most astrocytomas are not radiosensitive, and survival is usually only 3-6 years. Survival may be extended by reoperation. The cystic or solid cerebellar astrocytoma can often be completely excised, and recurrence is uncommon.

Medulloblastoma.

Medulloblastomas comprise about 10% of all gliomas. They occur almost exclusively in children 4-8 years of age, usually boys (3:1). They are composed of small, round, deeply staining cells, arise from the lateral wall or roof of the fourth ventricle, and frequently seed to other parts of the nervous system.

Complete surgical removal is not possible. These tumors are initially radiosensitive and literally "melt" with the first full course of treatment. However, they become radioresistant, and survival beyond 5 years is not common.

Ependymomas.

Ependymomas (about 10% of gliomas) arise most commonly in the wall and floor of the fourth ventricle, but can arise in the lateral ventricles.

Complete removal, particularly of those in the fourth ventricle, is rarely possible, and operative mortality is high. With subtotal removal, however, a survival span of 5 years is not uncommon. Ependymomas arising from the choroid plexus in the lateral ventricles may be totally removable.

Ependymomas are amenable to x-ray therapy.

Oligodendrogliomas.

Oligodendrogliomas (about 5% of gliomas) usually occur in adults. They are composed of uniform cells which have a "halo" around the nucleus and usually arise in the cerebral hemispheres.

Total removal is frequently not possible, and the cells are relatively radioresistant. Average survival is 3-7 years.

Pinealomas.

Pinealomas (2-3% of gliomas) usually occur in young adults and block the flow of CSF early. Surgical removal has an extremely high mortality rate. The treatment of choice is to shunt the CSF around the block (Torkildsen procedure) and administer x-ray therapy. The prognosis for survival is 3-10 years.

Miscellaneous Gliomas.

Astroblastoma, spongioblastoma polare, neuroepithelioma, ganglioneuroma.

MENINGIOMAS

Meningiomas arise from arachnoidal "cap cells" or from stromal cells of the pia arachnoid and dura. The course is usually prolonged and slowly progressive. Meningiomas occur predominantly in the 40-60 age group and frequently produce changes in the overlying bone. Since meningiomas involve the cerebral cortex early in their growth, convulsive seizures are often the first manifestation of their presence. Characteristic locations (in descending order of frequency) are as follows:

A. Parasagittal: Anterior parasagittal meningiomas produce predominantly headache and personality change. Mid-parasagittal meningiomas produce a classic picture of paresis of the contralateral foot with or without cortical sensory change, incontinence

of urine, and seizures beginning in the contralateral foot. Posterior parasagittal meningiomas produce headache and visual field defects.

B. Over the Convexity of the Cerebral Hemisphere: Symptoms depend upon the location, but in general these tumors are anteriorly situated and produce mental changes and motor power loss. Tumors in the mid area produce motor and sensory loss, and posteriorly situated tumors produce sensory and visual field losses.

C. Sphenoid Ridge: Meningiomas along the inner third of the sphenoid ridge cause extraocular muscle palsies and visual loss, with optic atrophy. Meningiomas along the middle third of the ridge usually attain a large size before significant symptoms are produced. Ultimate encroachment on the temporal lobe causes temporal lobe fits; encroachment into the frontal lobe produces mental changes. A meningioma occurring along the outer third of the ridge often grows as a flat plate of tumor (en plaque) and produces progressive exophthalmos and a palpable mass in the temporal region.

D. Olfactory Groove: These usually become quite large before clinical manifestations appear (anosmia and personality changes).

E. Less common locations are suprasellar, in the posterior fossa, and in the region of the gasserian ganglion.

Treatment.
　　Surgical removal is indicated in all cases. Meningiomas are benign tumors, and complete removal is often possible. Even with subtotal removal the prognosis is good.

TUMORS OF THE CRANIAL NERVES

　　Cranial nerve tumors arise from the sheath of the nerve (schwannomas, neurinomas, and perineural fibroblastomas). The great majority arise from the eighth cranial nerve at the internal auditory meatus (so-called acoustic tumors or cerebellopontine angle tumors).

　　Total surgical removal is indicated if possible. Where total removal is not possible (because the capsule is intimately adherent to the brain stem), the prognosis with subtotal removal may still be good since some of these tumors grow very slowly. Reoperation can be done when symptoms recur, but the operative mortality is much higher with the second operation.

TUMORS OF THE INTRACRANIAL BLOOD VESSELS

Hemangiomas.
　　Hemangiomas are actually only malformations with no neoplastic elements, e.g., capillary telangiectasia, which may be multiple and associated with telangiectasis of the skin (Sturge-Weber syndrome). They usually occur in the pons and are usually clinically silent, although occasionally they may produce symptoms if the malformation ruptures or obstructs the flow of CSF. Surgery is indicated only if CSF obstruction occurs with later signs, and consists

of bypassing the obstruction with a shunting procedure (ventriculocisternostomy; Torkildsen procedure).

Hemangioblastomas.

These are true neoplasms formed by proliferation of angioblasts. They are usually found in the cerebellum in adults. If there is associated hemangioblastoma of the retina, the disorder is called Lindau-von Hippel disease. Hemangioblastomas in the cerebellum frequently produce a large cyst filled with thick yellow fluid with the tumor represented in the wall of the cyst as a small mural nodule. Symptoms are those of a cerebellar lesion (see p. 515).

Surgical removal of the mural nodule is curative. Solid tumors are difficult to excise completely, and the prognosis is less favorable.

CONGENITAL INTRACRANIAL TUMORS

Craniopharyngioma (Rathke Pouch Cyst).

Craniopharyngiomas are the most common congenital brain tumors (3-4% of all brain tumors). They arise from epithelial cell rests in the region of the infundibular stalk, and consist of squamous epithelium or epithelium of the type seen in developing tooth buds (also called adamantinomas). The cyst may become quite large. Deposits of calcium above the sella turcica can often be seen by x-ray. Symptoms are produced by compression of neighboring structures, and consist of endocrine disorders, visual field defects, and optic atrophy. If the cyst encroaches on the third ventricle, increased intracranial pressure produces headache, nausea and vomiting, and papilledema.

Treatment is by surgical removal, although in the majority of instances total removal is not possible as the capsule is adherent to the hypothalamus or the carotid artery. Aspiration of the cyst and subtotal removal of the capsule carries a low operative mortality, but symptoms soon recur. The long-term prognosis is poor.

Epidermoid Cysts (Pearly Tumors, Cholesteatomas).

Epidermoid cysts arise from embryonic epidermal cells, and are composed of a glistening white fibrous capsule (looks like mother-of-pearl) and squamous epithelium; the center of the cyst is filled with grumous material and cholesterol crystals. Symptoms are usually delayed until adulthood. The cysts tend to occur near the midline.

Treatment is by complete surgical removal, when possible. If the wall is adherent to some vital structure, complete surgical removal is not possible, but long-term survivals have been reported even with incomplete removals.

Dermoids.

These resemble epidermoids but contain dermal structures, e.g., sebaceous glands and hair follicles. Treatment is as for epidermoid cyst.

Teratomas.

Teratomas are derived from 3 germinal layers and may contain

caricatures of the structures usually derived from these layers. Symptoms usually occur in childhood, and the lesion may attain huge size.

Where complete removal is possible the prognosis is good; the prognosis is poor if, as is more often the case, the tumor cannot be completely removed.

Chordomas.

These rare, slow-growing neoplasms are persistent remnants of primitive notochord which occur in characteristic location along the base of the sphenoid beneath the pons and extending into the middle fossa and cerebellopontine angle. They produce multiple cranial nerve palsies combined with long tract signs from the pressure on the pons. Marked erosion of the base of the skull is usually visible on basilar x-rays.

Total removal is rarely possible, and the prognosis is poor.

Paraphysial Cysts of the Third Ventricle.

These tumors, located in the anterior portion of the third ventricle and attached to the roof of the ventricle by a stalk, are thought to be derived from the paraphysis. They are cystic, smooth-walled, and lined by cuboidal epithelium. Symptoms are produced by intermittent blockage of flow of CSF through the foramen of Monro. Since the cyst hangs from a stalk, changes in body position may produce (or relieve) sudden violent headache.

Surgical removal is carried out by a transcortical approach to the lateral ventricle and then through the foramen of Monro to reach the third ventricle. The prognosis is good.

Pituitary Tumors.

Pituitary tumors arise from the anterior lobe and may be composed of any of 3 cell types: chromophobic, chromophilic or eosinophilic, and basophilic.

A. Chromophobe adenomas comprise the great majority of pituitary tumors. They are composed of chromophobe cells which have no known secretory function and therefore produce symptoms by compressing the secretory cells of the anterior pituitary; this causes signs of hypopituitarism: loss of libido, decreased cold tolerance, pale skin, and loss of axillary and pubic hair. As the tumor enlarges it balloons the sella turcica to give a characteristic x-ray enlargement and compresses the optic chiasm to produce bitemporal hemianopsia, usually the presenting symptom. The diagnosis can usually be made easily on the basis of the appearance of the patient and visual field and x-ray changes. Laboratory tests can be used to confirm the presence and degree of hypopituitarism.

The primary objective of therapy is to preserve vision, since endocrine changes are usually not reversible after radiation or surgical treatment. These tumors respond well to x-ray therapy, and in those cases where no visual field involvement is present radiation therapy is the treatment of choice. However, the visual fields should be checked daily during the course of radiation treatment; if the tumor swells and produces visual field defects, surgical removal of the tumor is indicated. Radiation therapy can be utilized postoperatively. In those

cases with slight visual field changes it may be difficult to
decide whether therapy should be by radiation or surgery. If
radiation is chosen, the visual field should be checked daily.

Total removal of these tumors is not often possible, but sub-
total removal and radiation therapy offer a very good prognosis.

B. Eosinophilic adenomas which develop before closure of the
epiphyses cause gigantism; those which develop in adult life
cause acromegaly. They rarely attain sufficient size to produce
significant visual field defects; in the great majority of cases
radiation therapy is the treatment of choice and offers a good
prognosis.

C. Basophilic adenomas rarely reach sufficient size to produce
local pressure symptoms, and are usually part of a polyglandu-
lar involvement producing Cushing's syndrome. They may be
removed by hypophysectomy.

Von Recklinghausen's Neurofibromatosis.

This is a heredofamilial disorder characterized by (1) café-au-
lait spots, (2) multiple neurofibromas on the peripheral, sympathetic,
spinal, and cranial nerves, and (3) multiple brain tumors of all
types: meningiomas, neurinomas, gliomas, etc. There is no effec-
tive therapy for the disease as a whole, but individual tumors caus-
ing disabling symptoms may have to be surgically removed. The
long-term prognosis is poor.

INTRASPINAL NEOPLASMS

Intraspinal neoplasms may occur at any level of the cord from
the foramen magnum to the sacral canal. The greatest number
occur in the thoracic area, as this is the longest subdivision of the
cord. They may arise from the spinal cord or its investing sheaths,
the nerves or their sheaths, bone, cartilage, fat, blood vessels,
fibrous tissue, or as metastatic lesions. Intraspinal neoplasms
may occur at any age, but are rare in persons under 10.

These tumors may be grouped for descriptive anatomic pur-
poses as follows: (1) Those arising within the spinal canal and ex-
tending extraspinally (or vice versa) through a vertebral foramen
(dumbbell tumors); (2) those arising within the spinal canal but not
invading the dura or cord (intraspinal extradural tumors); (3) those
arising within the dura but not invading the spinal cord (intradural
extramedullary tumors); and (4) those arising entirely within the
substance of the cord (intramedullary tumors).

In general, the same types of tumors are found in the spinal
canal as in the cranial vault, but the frequency of occurrence of
individual tumor types is quite different: neurofibromas, 31%;
meningiomas, 28%; intramedullary gliomas, 23%; and all others, 18%.

Since 60-70% of intraspinal tumors are benign, early diagnosis
and treatment are essential before irreparable spinal cord damage
has occurred.

Clinical Findings.

A history of pain in the back with radiation into dermatomal
patterns, accompanied by sensory and/or motor loss of a root or
long tract type, leads to a clinical diagnosis of spinal cord tumor.

When these symptoms are rapidly progressive, surgical treatment is urgent. This constitutes one of the most important emergencies in neurologic surgery.

A. **History:** One of the most important diagnostic findings is a history of more or less relentless progression. It is imperative to establish (1) whether symptoms began abruptly and have not changed significantly since onset, in which case an intraspinal tumor can be ruled out; (2) whether symptoms began abruptly and since that time have been marked by considerable exacerbation and intervals of remission, as one would expect in multiple sclerosis; or (3) whether the onset was vague but progression since onset has been more or less steady, as one would expect in spinal cord tumor.

B. **Symptoms and Signs:** One of the earliest symptoms of spinal cord tumor is radicular pain, i.e., pain produced by direct or indirect pressure or traction on the posterior (sensory) nerve roots. The pain is often experienced in a peripheral area of the dermatome supplied by the root or roots involved, e.g., tumors of the cervical region may cause pain in the shoulder, arm, or hand. In order to avoid an incorrect diagnosis of disease underlying the point of pain, the physician must secure a careful description of the type of pain. Radicular pain has the following rather distinct characteristics: (1) It is often restricted to the involved dermatomes; (2) it is severe, sharp, and stabbing, superimposed on a background of continuous dull, aching pain; and (3) it is made worse by coughing, sneezing, or straining, worse at night after prolonged recumbency, and temporarily relieved by activity. Paresthesias and/or dysesthesias are variously described as numbness, coldness, tingling, and hot or crawling sensations, also in a radicular pattern.

The sensory loss may follow a root pattern at the level at which the roots are involved by the tumor; and the sensory loss below this level, if present, is of a spinal cord type, i.e., it produces a distinct level of sensory loss with often greater involvement of 1 modality of sensation than another. Motor involvement may be of the upper motor neuron type, i.e., spasticity, hyperreflexia, and extensor plantar response; or of the lower motor neuron type if the tumor involves the cauda equina.

Hesitancy in urination is a frequent early sign of pressure on the spinal cord.

C. **X-ray Findings:** X-rays of the spine frequently give additional evidence of the presence of a tumor, i.e., erosion, calcium deposition in the tumor, increase in the interpedicular distances, enlargement of the intervertebral foramens, etc. The diagnosis can be confirmed by positive contrast myelography (see p. 510).

Caution: If in carrying out the preliminary spinal tap a total manometric block is discovered and a block is found to the flow of the contrast material, no attempt should be made to remove the medium since removal of spinal fluid from below the block may lead to "jamming" or descent of the tumor and immediately increase the neurologic deficit.

Treatment and Prognosis.

Surgical exploration and excision is indicated in every case.

Most benign tumors can be removed completely. The prognosis for return of function depends upon the location of the tumor, whether it can be removed completely, and the severity and duration of neurologic deficit prior to diagnosis. The prognosis in intramedullary tumors is more guarded, as the hazard of increasing the neurologic deficit rarely permits complete removal. Postoperative x-ray therapy may be beneficial in diminishing the mass of these tumors and preventing further neurologic losses.

METASTATIC INTRASPINAL TUMORS

The spinal cord is not infrequently involved secondarily by metastases to the bones of the spinal column or to the epidural space. These lesions produce secondary compression of the spinal cord and paraplegia. When obvious compression of the spinal cord occurs, laminectomy and decompression should be seriously considered. The patient's general condition and life expectancy, taking into account other metastases and the type and location of the primary tumor, may contraindicate laminectomy. In general, if the patient's life expectancy is felt to be at least 6 months and his general condition is good, decompressive laminectomy is indicated.

PERIPHERAL NERVE TUMORS

Perineural Fibroblastomas.
The most common tumors of the peripheral nerves are those arising in supporting tissue (perineural fibroblastomas). They are usually solitary, well encapsulated, and benign, and may be of any size. Diagnosis is usually suggested by a mass in the periphery. Symptoms include paresthesia or hypesthesia; with tumors of mixed or pure sensory nerves, percussion over the tumor frequently elicits tingling (Tinel's sign). Malignant change (sarcoma) occurs only rarely.

Treatment is by surgical removal. In many cases it is relatively easy to remove the tumor completely from its capsule; in other instances removal may be quite difficult and may cause permanent impairment of function.

Neurofibromas.
Neurofibromas are composed of all the elements of peripheral nerves (axis cylinders, myelin sheaths, and connective tissue). These may appear as solitary lesions but most commonly are multiple, occurring as part of von Recklinghausen's neurofibromatosis. Multiple excision is rarely indicated, as these tumors cannot be shelled out and surgical removal requires interruption of the continuity of the nerve trunk.

Sarcomatous change (neurogenic sarcoma) is more common in neurofibromas than in sheath tumors. Although neurogenic sarcoma may not be distinguishable clinically from the benign tumors except for the rapidity of its growth, at surgery its sarcomatous characteristics are demonstrated by infiltration of surrounding structures. Distant metastases also occur.

CEREBROVASCULAR DISEASE

Surgical disorders of the cerebral blood vessels produce their clinical effects by 1 of 2 pathologic processes: (1) Occlusion, leading to ischemia, and (2) hemorrhage.

CEREBRAL ISCHEMIA

Any area of the brain may be involved by an ischemic process produced by (1) partial occlusion of an extracranial or intracranial vessel as a result of narrowing of the vessel by atheromatous plaques and/or progressive thrombus formation on these plaques, (2) complete occlusion, usually at the site of atheromatous plaques; or (3) by embolization. These processes occur most commonly at the point of bifurcation of the common carotid artery into the internal and external carotid arteries; at the bifurcation of the internal carotid artery into the middle and anterior cerebral arteries; at the point of origin of the common carotid artery from the aortic arch or innominate artery; and at the point of origin of the vertebral artery. Symptoms are often produced by each new deposition of thrombus as the lumen closes or by episodes of relative hypotension. In the early stages, these attacks are frequently transient and have been termed "little strokes." Symptoms may develop slowly but progressively via hours or days where there is a gradual thrombosis formation, or there may be a sudden and catastrophic onset of symptoms produced by embolization or sudden complete occlusion of the vessel.

Recent studies have indicated that about 15-20% of all cerebrovascular accidents are due to occlusive processes, either incomplete or complete, in the major extracranial vessels in the neck. Because these stenotic lesions are sometimes amenable to surgical attack, diagnosis should be made as early as possible before permanent residuals have occurred.

Clinical Findings.

The diagnosis is suggested by (1) a history of any number of transient "little strokes"; (2) decreased pulsation of either carotid artery in the neck; (3) the presence of a bruit over either carotid bifurcation or over the take-off of the carotid and vertebral arteries low in the neck; and (4) symptoms referable to the area of the brain supplied by the vessels (see below). Ophthalmodynamometry (measurement of intraocular arterial pressure) may be diagnostic if the pressure in the retinal artery on the involved side is significantly lower than that on the other side. Cerebral arteriography is essential to establish the diagnosis.

  A. Internal Carotid Artery: Occlusion usually produces contralateral weakness, subjective numbness, ipsilateral blindness (from involvement of the ipsilateral ophthalmic artery in the temporary ischemic process), and, if the predominant hemisphere is involved, aphasia.

  B. Middle Cerebral Artery: Usually the same symptomatology as total internal carotid insufficiency, but no blindness.

  C. Anterior Cerebral Artery: Weakness and/or subjective

numbness of the contralateral leg and occasionally the arm, and, not infrequently, reflex incontinence.

D. **Posterior Cerebral Artery:** Hemianopsia, scintillating scotomas, and, if bilateral, temporary cortical blindness.

E. **Basilar Artery:** Usually bilateral symptoms such as quadriparesis, bilateral paresthesias, ataxia, dysarthria, diplopia, dysphagia, blindness, and frequently unconsciousness.

Treatment.

There is no effective treatment for complete occlusions and no effective surgical treatment for intracranial involvement at the branches of the carotid or vertebral systems.

A. **Medical Treatment:** Long-term anticoagulant therapy in the hope of preventing further thrombus formation.

B. **Surgical Treatment:** In partial occlusions of the extracranial portions of the carotid or vertebral arterial systems, surgical therapy is aimed at restoring the blood flow to normal by thromboendarterectomy or bypass procedures. Angiograms should be taken on both sides preoperatively to demonstrate the patency of the carotid system. Visualization of the aortic arch may also be necessary.

CEREBRAL HEMORRHAGE

Spontaneous intracranial hemorrhage may be caused by hypertension, rupture of an intracranial aneurysm, rupture of an arteriovenous malformation, or hemorrhagic disorders. Of these, only hemorrhages due to aneurysms and arteriovenous malformations are of surgical importance. The hemorrhage may be subarachnoid (most common), intracerebral, or subdural (rare).

INTRACRANIAL ANEURYSMS

Most intracranial aneurysms are congenital and most occur on the anterior portion of the circle of Willis, usually at the point of bifurcation of the major arteries (e.g., carotid–posterior communicating). About 10-15% occur in branches of the basilar artery, including the posterior cerebral arteries. In approximately 15% of cases multiple aneurysms are present.

Clinical Findings.

Intracranial aneurysms produce symptomatology by 1 or all of the following processes: (1) Subarachnoid hemorrhage and, less frequently, intracerebral and subdural hemorrhage; (2) pressure on nearby structures, which most commonly produces cranial nerve palsies (third, fifth, second, fourth, and sixth) but may produce symptoms due to ischemia if the aneurysm partially occludes neighboring or parent arteries; and (3) distention of or pressure upon pain-sensitive structures, producing headache, usually orbital or supraorbital.

In subarachnoid hemorrhage due to rupture of an intracranial aneurysm, the mortality with the first episode of bleeding is 30-40%. These most commonly occur as a catastrophic illness in previously

healthy individuals 20-40 years of age. The danger of a second
hemorrhage is greatest within the first 3 weeks after the initial
episode of bleeding. The mortality rate increases with successive
hemorrhages. A sudden onset of severe headache with or without
loss of consciousness is followed by signs of severe meningeal irri-
tation (stiff neck, photophobia, diffuse headache, irritability, etc.).
The diagnosis is established by lumbar puncture, which reveals
homogeneously bloody fluid (see p. 491), usually under markedly
increased pressure. The aneurysm may also rupture into the brain
substance (intracerebral) and thereby produce profound neurologic
loss (e.g., hemiplegia, aphasia), coma, or death.

Arteriograms should be taken immediately or as soon as the
patient's condition permits.

Treatment.
  A. General Measures: Absolute bed rest, maintenance of fluid in-
     take, and mild analgesics to control headache and restlessness.
  B. Surgical Measures: If surgical treatment is indicated it should
     be done as soon as the patient's condition permits.
     1. Direct intracranial approach - Treatment is aimed at iso-
        lating the aneurysm from the circulation (without producing
        a neurologic deficit) by (1) clipping the neck of the aneurysm
        or (2) trapping the aneurysm without compromising the distal
        circulation of the parent vessel. For example, aneurysm of
        the anterior cerebral artery proximal to the communicating
        artery can be trapped by clipping the anterior cerebral ar-
        tery proximal and distal to the aneurysm, thus leaving the
        distal anterior cerebral arterial supply intact by virtue of
        the blood crossing over through the anterior communicating
        artery. Direct intracranial surgical attack on these lesions
        has been greatly facilitated by the use of hypothermia.
     2. Indirect surgical approach - The purpose of ligation of the
        carotid artery in the neck is to reduce the intraluminal
        pressure in the aneurysm and thus reduce the danger of sub-
        sequent hemorrhage. In general, this method of treatment
        is only applicable to aneurysms arising from the internal
        carotid artery below the circle of Willis, since the collateral
        circulation from the contralateral carotid artery will main-
        tain high intraluminal pressure in aneurysms above the
        circle.

CEREBRAL ANGIOMAS

Cerebral angiomas are congenital lesions which may cause (1)
subarachnoid or intracerebral hemorrhage (hemorrhage is far less
common in angioma than in aneurysm); (2) recurrent convulsive
seizures; (3) loss of function of adjacent brain, producing neuro-
logic deficits such as hemiparesis and aphasia; or (4) progressive
mental deterioration. Neurologic deficit may be due to hemorrhage,
with subsequent gliosis and cyst formation; or shunting of the blood
supply away from the brain tissue directly into the venous system,
with resultant hypoxia, gliosis, etc.

The diagnosis is suggested by the history, the presence of an
audible bruit over the lesion, and intracranial calcification on x-ray.
Cerebral angiography is diagnostic.

The only effective method of treatment is surgical removal. The prognosis depends upon the size and site of the lesion (superficial or deep, in the dominant or nondominant hemisphere) and the presence and severity of the preoperative neurologic deficit.

INFECTIONS OF SCALP, BONE, AND BRAIN

PYOGENIC SCALP INFECTIONS

All pyogenic infections of the scalp should be treated vigorously, as there are abundant communications via the venous channels from the scalp to the diploic spaces of the calvaria and from these diploic spaces to the underlying dura, etc. Early and vigorous treatment of scalp infections by warm soaks and antibiotics should be employed to prevent the spread of infection to these deeper structures.

OSTEOMYELITIS OF THE SKULL

Osteomyelitis of the skull occurs most commonly (1) by direct implantation of bacteria into bone by trauma, or (2) by direct extension from infection in contiguous structures, e.g., scalp or sinuses.

A recent or remote history of head trauma or of infection in a nearby structure can usually be obtained, although onset of signs and symptoms may be delayed for a considerable period after the initial insult. Typical manifestations are headache, local evidence of inflammation around the involved area (with or without a draining sinus), and pain upon palpation of the involved area. Generalized systemic effects such as fever, leukocytosis, cervical or suboccipital lymphadenopathy, etc., may not be present. X-rays may be negative early, but later reveal the characteristic mottling or moth-eaten appearance.

Most cases of osteomyelitis can be prevented by proper debridement, sterile technic, and adequate initial therapy in trauma and by prompt treatment of infection of adjacent structures. Vigorous antibiotic therapy is indicated, and should be based on cultures and sensitivity tests. The original infection should also receive appropriate therapy. If the process continues to advance despite antibiotic therapy, all infected bone and a generous margin of normal bone should be excised. Direct instillation of the appropriate antibiotic may be helpful. Cranioplasty should be deferred until at least 6-12 months after all evidence of infection has disappeared.

EPIDURAL ABSCESS

Epidural abscess usually results from direct extension of an overlying infection, although hematogenous spread does occur. The classical findings in cranial epidural abscess are those of a rapidly enlarging space-occupying lesion in association with the symptoms and signs of systemic infection. Spinal abscesses cause sudden, excruciating pain accompanied by rapidly advancing motor paralysis of all functions below the level of the lesion.

This is a critical surgical emergency. In intracranial

epidural abscess multiple bur holes are placed to effect complete drainage of all loculated areas. (Adherence of the dura to the suture lines may localize the abscess to a specific area.)

Appropriate antibiotics are given systemically and directly into the epidural space. In the spinal canal the abscess should be completely evacuated and the wound packed open and allowed to granulate in.

The prognosis for life and for the return of lost neurologic function depend upon the virulence of the infective organism and the promptness and vigor of surgical treatment.

SUBDURAL ABSCESS

Subdural abscess is usually the result of direct extension of infection from an overlying area, but may occur following rupture of an intracranial abscess into the subdural space. Since there are no limiting structures in the subdural space these abscesses extend over the entire hemisphere, under the brain, and into the interhemispheral fissure.

The clinical findings are those of a rapidly enlarging space-consuming lesion: lethargy, obtundation, paresis, coma, papilledema, etc. The diagnosis is based upon a history of primary infection and the finding of subdural pus on bur hole exploration.

Treatment is by placement of multiple bur holes so that every portion of subdural space over the hemisphere and in the interhemispheral fissure can be adequately drained. Massive systemic antibiotic therapy is indicated as well as direct instillation of antibiotic into multiple small polyethylene catheters left in the subdural space. The mortality is high.

INTRACEREBRAL ABSCESS

A variety of bacterial organisms as well as animal parasites, yeasts, fungi, and molds may cause intracerebral abscess. Eight out of 10 cases are due to direct extension of infection from the mastoid bone, paranasal sinuses, or middle ear. These cavities are expanded diploic spaces and so have thinned out inner and outer walls that offer slight resistance to the spread of infection. Abscesses caused by direct extension are usually single, whereas the 20% arising from distant sources by hematogenous spread are usually multiple. Half of those which are metastatic from distant foci originate in the lung (bronchiectasis or lung abscess).

Most abscesses begin in the area of least vascularity, about 10-20 mm. below the cortex. In those that originate by direct extension the infectious process incites an osteomyelitis which extends by suppurative retrograde thrombophlebitis along the diploic veins to involve the dura. The underlying arachnoid becomes involved in the inflammatory process and becomes sealed off to prevent lateral spread; the infectious process thus proceeds into brain tissue and the intracerebral abscess forms. During the initial acute stage, the infection is not well sealed off by the brain and cerebritis results. As the process becomes localized, a pseudocapsule develops and gradually is transformed into a true capsule composed of astrocytes

and fibroblasts. The surrounding cerebritis and edema subside, and the abscess becomes a chronic one, acting as a space-consuming lesion.

Clinical Findings.

A history of infection of the ears or sinuses, or of a systemic infection, is most important. In a typical case the signs of the original infection in those areas may begin to subside only to be followed by convulsions, increasing headache, and paresis, with progressive lethargy. In the acute phase the patient is toxic with fever, leukocytosis, and an elevated CSF cell count. In the chronic phase, as the process is walled off by the capsule, the toxic symptoms are replaced by signs of a space-consuming lesion. Lumbar puncture may reveal elevation of cell count and protein, but should not be done in the presence of papilledema.

The diagnosis is confirmed by air studies or arteriography. Because the present widespread indiscriminate use of antibiotics frequently masks the characteristic progression of signs and symptoms from the acute through the chronic phases, the possibility of intracerebral abscess must always be kept in mind.

Treatment.

In the acute phase massive treatment with appropriate antibiotics must be given promptly. In the subacute stage, if suppuration is occurring and the infection is well localized, the abscess cavity can be tapped through a bur hole, the pus aspirated, 1-2 ml. of thorium dioxide (Thorotrast®) instilled into the cavity. As the macrophages in the wall of the abscess begin to take up the medium, the exact size and location of the abscess can be seen by x-ray, if subsequent tapping or direct instillation of antibiotics is required, the area can thus be accurately cannulated. The abscess may have to be tapped several times during convalescence.

After recovery the abscess wall should be resected if this will not produce a neurologic deficit. In chronic, well-encapsulated abscess, total excision is the treatment of choice.

PAIN

The neurosurgeon is frequently called upon to carry out a pain-relieving surgical procedure for the purpose of relieving chronic pain of known or unknown origin. Every effort must be made to determine the cause of the pain, and every patient who complains of chronic pain which does not have an apparent cause or does not fall into a well-established clinical category must be thoroughly evaluated from the psychiatric point of view before surgery is considered.

The most common painful syndromes requiring surgical pain-relieving procedures are trigeminal neuralgia, glossopharyngeal neuralgia, post-herpetic neuralgia, and pain produced by advanced malignancies.

TRIGEMINAL NEURALGIA
(Tic Douloureux, Trifacial Neuralgia)

The etiology of trigeminal neuralgia is not known. It occurs most commonly in the age group from 40-60.

The type of pain in trigeminal neuralgia is characteristic. It occurs without warning in the distribution of any of the major branches of the trigeminal nerve, and is severe, lancinating, "bright" pain. Each pain is a brief short jab, but attacks frequently come in flurries.

The third division of the fifth cranial nerve is most commonly involved. The paroxysms of pain are most frequent in the spring and fall, and there may be periods of remission lasting for many months. Most patients soon discover that there is an apparent "trigger" area somewhere about the face, mouth, or tongue. When this area is stimulated by touch, chewing, or a cold breeze, the paroxysm results. These patients understandably go to great lengths to avoid stimulating this "trigger" area and may refuse to eat, shave, talk, etc. It is imperative to remember that in true trigeminal neuralgia the pain is not steady or aching, there is no pain between attacks, and there is no loss of sensation in any division of the trigeminal nerve. If there is a demonstrable loss of sensation over the trigeminal distribution, the pain is more likely caused by a tumor in or near the gasserian ganglion.

Treatment.

In the vast majority of cases of true tic douloureux, the pain can be permanently relieved by section of the sensory root of the fifth nerve. However, this leaves the face on the side of the operation permanently anesthetized, and since the cornea on that side is also anesthetized the risk of keratitis must be considered. Surgery should therefore be done only as a last resort and more conservative measures tried first.

A. Conservative Measures:
1. Medical treatment - Diphenylhydantoin sodium (Dilantin Sodium®), 100 mg. (1½ gr.) t. i. d. - q. i. d. for at least 2 weeks, should be given a trial in all cases, since a significant number of patients report at least temporary relief on this regimen.
2. Alcohol block - If diphenylhydantoin is ineffective, alcohol blocks of the involved division or divisions of the trigeminal nerve should be carried out. The mandibular division is blocked at the foramen ovale, the maxillary division at the foramen rotundum; the only part of the first or ophthalmic division which can be blocked is the supraorbital nerve where it exits in the supraorbital ridge. Alcohol block renders the nerve functionless for varying periods. Relief following the first block frequently lasts 12-24 months, but with each succeeding block the scarring produced about the nerve makes it more difficult to obtain a good block, and relief may last several months or there may be no relief. After 2 alcohol blocks the patient should be advised to undergo surgery.
B. Surgical Measures: The surgical treatment of choice is intracranial section of the sensory root of the trigeminal nerve through the subtemporal approach. Some surgeons advocate

compression of the ganglion, exposing and vigorously rubbing but not cutting the ganglion and posterior root; it has been found that this relieves the pain in a significant number of patients and prevents facial and corneal anesthesia. If pain recurs, sectioning of the root is indicated. If the pain is limited to the second and third divisions, subtotal section of the sensory root can be carried out in an attempt to preserve the fibers carrying sensation to the cornea. The prognosis is excellent; in true tic douloureux, complete relief can be obtained in over 95% of cases.

GLOSSOPHARYNGEAL NEURALGIA

The pain of glossopharyngeal neuralgia is of the same type as described for trigeminal neuralgia, but is located in the distribution of the ninth cranial nerve, in the tonsillar fossa, and deep in the neck at the angle of the jaw on the affected side. The diagnosis can be easily confirmed by cocainizing the tonsillar fossa on the affected side; in true tic douloureux this immediately relieves the pain, and pain recurs after the local anesthetic has worn off. Intracranial section of the ninth and the upper 2 filaments of the tenth cranial nerves through a posterior fossa approach gives permanent relief. The prognosis is excellent.

POST-HERPETIC NEURALGIA

In a few cases of herpes zoster there is a persistent severe burning pain in the involved area after the infection has run its course. Chronic inflammatory changes have been shown to be present not only in the posterior root ganglion but also in the ascending pathways in the spinal cord and brain stem carrying the pain impulses. Because these changes are so widespread, surgical procedures designed to interrupt the pain-conducting pathways at a particular level are seldom successful. Relief has been obtained by undermining the skin in a wide margin around the involved area. (If the ophthalmic division of the fifth cranial nerve is involved, a large skin flap is elevated; if an intercostal or other area of the body is involved, the entire area is undercut.) In other cases relief has been obtained by various peripheral neurectomies. Because of the widespread inflammatory changes in the pain-conducting pathways, 'the simplest procedures should be tried first before resorting to cordotomy or tractotomy in the brain stem; and it may be necessary, as a last resort, to perform a restricted frontal leukotomy.

PAIN PRODUCED BY ADVANCED MALIGNANCIES

Severe pain becomes a problem as inoperable malignancies invade pain-sensitive structures. The pain produced is usually deep, boring, and steady, with sharp, stabbing pains at times superimposed. Relief can be given without loss of vital neurologic function by cutting the fibers responsible for pain transmission at

an appropriate level in the peripheral or central nervous system.

Operations for relief of pain in cancer should not be thought of only as a last resort. As soon as chronic pain becomes a problem in operable malignancy, the patient should be evaluated as a candidate for pain-relieving surgery and a decision reached regarding which procedure offers the best chance for success. Large doses of narcotics over prolonged periods create the problem of addiction and induce an undesirable mental dullness and disinterest in life. As a general rule, if, in the clinician's opinion, the patient's life expectancy is longer than 3-6 months, a pain-relieving surgical procedure should be carried out as soon as the pain becomes significant.

The most frequently utilized operation is a cordotomy (spinothalamic tractotomy); the spinothalamic tract in the anterior quadrant of the spinal cord on the side opposite the pain is sectioned at the high thoracic or high cervical level. The patient thus loses the ability to perceive pain and thermal sensation on the contralateral side of the body below the level of the section. For purely unilateral pain involving the midline area, or for bilateral pain, bilateral cordotomy on the contralateral side is all that is required. For pelvic pain involving the midline area, or for bilateral pain, bilateral cordotomy is indicated. For relief of any pain below the level of the xyphoid, a thoracic cordotomy at the T1-T3 level may suffice. To relieve pain in the upper thorax or arm, the cordotomy should be carried out at the C1-C2 level; this "high cervical cordotomy" is favored by many neurosurgeons as the procedure of choice regardless of the level of pain. In pain involving the face, jaw, neck, or brachial plexus the pain fibers can be sectioned in the medulla at the level of the obex. An alternative procedure is to section the trigeminal, glossopharyngeal, and upper filaments of the vagus nerves intracranially and the upper 3 or 4 posterior cervical roots on the side of the pain. In rare instances where more caudad operations have failed to give satisfactory relief, a unilateral frontal leukotomy (medial) may be considered.

Pain transmission can be interrupted at the thalamic level by electrocoagulation of the thalamic nuclei responsible for pain conduction, utilizing a stereotaxic apparatus (see below).

STEREOTAXIC SURGERY

Within the past several years it has become possible to destroy various nuclear masses and fiber tracts deep within the brain with a high degree of accuracy by utilizing a human stereotaxic apparatus. This consists of a rigid metal frame which can be temporarily firmly affixed to the patient's head by metal pins. Mounted on this frame is an electrode carrier. The electrode can then be inserted into the brain through a properly placed bur hole to any desired depth at any angle. Lesions can then be produced by electrocoagulation and can be graduated to any size. The nuclear mass or fiber tract selected as the target is localized by use of a coordinate

system derived from cadaver material, x-rays, and pneumoen-cephalography.

The field of stereotaxic surgery is relatively new, and only a few diseases are now being treated in this fashion; but this technic holds great promise for a wider scope of application in the future. The results to date are much better than those obtainable in the past with older technics.

At present, the major application of this technic is in the following conditions:

A. Hyperkinetic movement disorders, e.g., as in parkinsonism, dystonia musculorum deformans, and choreo-athetosis. Lesions are placed in the medial globus pallidus and/or the ventrolateral nucleus of the thalamus on the side of the brain opposite the side of the involved extremities. This operation can be carried out bilaterally if there are severe bilateral symptoms. Surprisingly effective relief can be obtained from the tremor and rigidity in parkinsonism and from the hyperkinesias in dystonia musculorum deformans and choreo-athetosis in properly selected cases.

B. Pain Problems: In cases of advanced inoperable malignancy where the pain is too high to be relieved by standard pain-relieving technics or if more caudad interruptions of pain pathways have failed to give sufficient relief, selective destruction of the nuclear masses within the thalamus concerned with pain transmission can be carried out with beneficial results.

19 . . .

Traumatic Orthopedic Surgery

FRACTURES AND DISLOCATIONS OF THE SPINE

Traumatic injuries to the spine most commonly result from indirect violence. Direct trauma is more likely to cause fracture of the spinous process, and rarely of the lamina. Open fractures are usually due to penetrating injuries. Fractures of the spine may be classified according to their anatomic location (i.e., body, pedicle, lamina, or muscular process), but classification according to regional location is more useful since neurologic deficit resulting from cord injury depends upon the level at which the injury occurs. Spinal injuries are thus classified here as cervical, thoracic, lumbar, sacral, or coccygeal.

In most cases of injury to the spine neither the spinal cord nor the nerve root is injured, but the possibility of such injury, especially in the cervical spine, is always present. Injury to the cord or nerve roots may result from displacement of bone fragments or spinal segments. Neurologic injury may occur at the time of injury or may be the result of subsequent manipulation. Since initial x-rays do not necessarily reflect the degree of displacement which may have occurred, severe neurologic deficit may be present even though only a minor fracture can be demonstrated. The greatest permanent disability caused by injury to the spine is due to associated injury to the spinal cord.

FRACTURES AND DISLOCATIONS OF THE CERVICAL SPINE

Concomitant injury to the cord or nerve roots can be expected in about 25% of cases of severe injury to the cervical spine. Fracture of the vertebral body, posterior displacement of fragments of the intervertebral disk, and dislocation account for most cord or nerve root injuries.

The diagnosis is suggested by a history of injury to the cervical spinal region and confirmed by careful physical examination and x-rays. Care must be taken to avoid manipulation of the neck. If routine films do not confirm the suspected injury, special studies may be required. An accurate diagnosis is essential before treatment is instituted.

Compression Fracture of the Cervical Vertebral Body Without Dislocation.

In lateral x-rays compression fracture of the superior plate of the vertebral body without apparent forward dislocation of the segment above is apt to be demonstrated by a wedge-shaped deformity with the apex directed anteriorly. Although films may demonstrate

no evidence of dislocation, the possibility of a prior dislocation and spontaneous reduction should be considered.

Compression Fracture of the Cervical Vertebral Body With Dislocation.

Complete anterior dislocation of the cervical spine is observed most commonly with fracture, but may occur without fracture (see below). Compression of the superior plate of the body, with disruption of the intervertebral disk, dislocation of the facet joints, and tearing of the posterior ligaments, accounts for the major lesions. If the facet joints do not dislocate, fracture of the pedicles can permit dislocation.

The lesion can be corrected by unsustained traction, but the deformity tends to recur when the force is discontinued. Continuous traction for 3-6 weeks is usually necessary before application of plaster or a brace. Because of the complications and discomfort associated with the use of head halter traction, skeletal traction is the method of choice until the deformity has been reduced and stability attained.

Anterior Dislocation of the Cervical Spine.

Partial or complete anterior dislocation of the cervical spine may follow acute flexion injury to the neck. Although bone injury may not be present, disruption of the intervertebral disk and tearing of the posterior interspinous ligaments and ligamentum flavum may occur and allow complete dislocation.

Complete dislocation occurs when the inferior articular processes of the dislocated segment are displaced anterior to the superior articular process of the segment below. It is likely to be associated with cord injury. If the articular processes are locked, reduction is difficult even with skeletal traction.

If the inferior articular processes of the segment are displaced forward but the articular surfaces of the facets remain in partial contact, the dislocation is termed incomplete, or a subluxation. It must be emphasized that x-rays may demonstrate only subluxation in cases where more extensive displacement has occurred but has partially reduced spontaneously since the injury. Any evidence of neurologic deficit should suggest that possibility.

Complete Unilateral Dislocation of the Cervical Spine.

Complete unilateral dislocation is characterized by torsional displacement in which the anatomic relationship of 1 facet joint remains undisturbed. Closed methods of reduction may not be effective when dislocation is complete. Closed manipulation is hazardous and should be attempted only by a specialist. If skeletal traction is not successful, open reduction is indicated. Complete unilateral dislocation is commonly the result of severe injury, and cord damage should always be suspected.

Unilateral Subluxation of the Cervical Spine.

In adolescents unilateral cervical subluxation may occur spontaneously during sleep without appreciable trauma. Rotation of the head and lateral flexion of the neck toward the affected side are restricted, so that the patient holds his head tilted toward the opposite side. The lesion is best demonstrated by lateral stereoroentgenography. Reduction can usually be accomplished by gentle manual

traction or by head halter traction using 5 lb. of weight for 24-48 hours. For a few days after reduction the neck should be immobilized and protected by a felt collar.

Atlanto-axial Dislocation in Children.

Spontaneous dislocation of the atlas and axis may occur as a complication of upper respiratory tract infection in children. Initial treatment should be by head halter traction; after the patient becomes ambulatory, bracing may be required.

Posterior Dislocation of the Cervical Spine.

Posterior dislocation is a rare lesion which may be due to a blow on the forehead causing sudden, acute hyperextension of the neck. It is usually associated with severe spinal cord injury.

Comminuted Fracture of the Cervical Vertebral Body.

Comminuted fracture of a cervical vertebral body may occur as a result of a heavy blow on the top of the head which drives the intervertebral disks above and below into the body, causing it to burst. A posterior fragment may impinge upon the spinal cord and cause severe injury. Skull traction is indicated until stabilization takes place. The head is then immobilized in a plaster Minerva jacket.

Fractures and Fracture-Dislocations of the Atlas and Axis (Epistropheus).

The most common injury involving the first and second cervical vertebrae is fracture of the odontoid process, with anterior dislocation of the atlas on the axis. The odontoid process is carried forward with the atlas, and this at least partially protects the cord from being crushed. Reduction can generally be accomplished by skull traction, but plaster immobilization must be maintained for at least 16 weeks since bony healing of the odontoid process is slow.

Bursting fracture of the atlas may also result from a blow on the top of the head. Displacement of the lateral mass takes place when the condyles of the occiput are driven into the atlas. If the spinal cord has not been injured and the fragments are not widely displaced, plaster immobilization for 12-16 weeks may be all that is required. Otherwise, skull traction for 4-8 weeks is required before immobilization.

Sprains of the Cervical Spine

When the normal range of motion is exceeded suddenly, a sprain of the cervical spine may result. The prominent clinical° feature of this poorly understood lesion is dull, aching pain in the back of the neck. Cervical sprain results most commonly when a stationary or slow-moving vehicle is struck suddenly from behind. Pain may not be felt until a few hours after the injury has occurred, and is associated with muscle guarding and restriction of movement. The pain may radiate into the occipital or the interscapular region. Posterior cervical tenderness is usually diffuse, and the site of pain poorly defined. If routine anteroposterior and lateral x-rays have been found to be negative, lateral flexion and extension films should be taken to determine the presence or absence of instability. Patients with degenerative arthritis which preexisted the injury

may demonstrate restriction of motion in the lower cervical segments. Where litigation or emotional lability are not factors, symptoms generally respond promptly with conservative measures including immobilization with a cervical collar, heat, massage, and analgesics.

Reduction of Injuries to the Cervical Spine.

A. Skeletal Traction: The head halter is useful for the application of heavy traction for short periods, as may be required during manipulative reduction. In general, the head halter is not suitable for continuous traction because more than 7-8 lb. causes pain in the region of the chin or occiput. Skeletal traction applied to the skull or zygomatic arches will support 40 lb. of force over prolonged periods without discomfort. Various types of tongs (e.g., those of Barton, Crutchfield, or Roger Anderson) and wires (Hoen) have been designed for this purpose.

After the skeletal apparatus has been applied, reduction is accomplished by increasing the force of traction under periodic x-ray control. Once reduction has been achieved, the force is gradually reduced to 8-12 lb., which is usually sufficient to maintain reduction.

If closed reduction is not successful, open reduction must be resorted to.

B. Open Reduction and Arthrodesis: When reduction cannot be accomplished by closed methods, open reduction may be successful. If open reduction is required it should be done early, especially if symptoms of cord injury are present.

FRACTURES OF THE THORACIC SPINE

The thoracic spine is comparatively stable. Fracture results either from direct violence, which may involve a spinous process; or indirect violence, which may result in compression of the body of the vertebra. Occasionally an avulsion fracture of the spinous process of the seventh cervical or first thoracic vertebra is caused by muscular activity ("clay-shoveler's" fracture)

Compression fractures of the thoracic vertebrae are rare in young children and are caused only by severe trauma in older children. In this age group, therefore, unless there is a positive history of severe trauma, a wedge-shaped deformity in the thoracic spine should suggest pathologic fracture. This must not be confused with Calvé's or Scheuermann's disease or deformity due to traumatic fracture.

Minimal trauma may cause compression fracture of the body of the thoracic vertebrae in adults with osteoporosis. Disability is not great, and reduction is not indicated. If rest in bed for 3-4 days does not relieve pain, a surgical corset or brace may be provided to permit early ambulation.

Compression fractures of the thoracic spine due to severe trauma are characterized by wedge-shaped deformity. No adequate method has been devised for the reduction of these injuries. However, because of the inherent stability of the thoracic spine, prolonged immobilization is not necessary. In fracture of the upper thoracic region, if immobilization is required for relief of pain, a

plaster Minerva jacket may be applied. This is usually necessary only for injuries of the upper thoracic region.

FRACTURES AND FRACTURE-DISLOCATIONS OF THE LUMBAR SPINE

Fractures of the Lumbar Spine.

A. Simple Compression Fractures: Compression fractures of the vertebral bodies caused by hyperflexion injury are the most common fractures of the lumbar spine and occur most often near the thoracolumbar junction. More than 1 vertebral body is often involved, but deformity may be greatest in 1 segment. Acute angulation of the spine caused by a compression deformity of the body of a vertebra may be associated with varying degrees of disruption of the facet joints, from sprain to complete dislocation.

Anteroposterior, lateral, and oblique x-rays are required to demonstrate the characteristic wedge-shaped deformity and the presence of dislocation of the facets, comminuted fracture of the body, and fracture of the pedicle.

Treatment depends upon the age of the patient, the presence of preexisting disease, and the severity of injury. In older patients with preexisting degenerative arthritis where there is mild deformity involving no more than one-fourth of the anterior height of the body of the vertebra, the surgeon may elect not to reduce the deformity but merely to place the patient at bed rest for a few days, after which the back should be braced. In the more active age group when the compression deformity involves more than one-fourth of the anterior height of the body of the vertebra, reduction by hyperextension and immobilization in a plaster jacket (see p. 540) is the treatment of choice.

B. Comminuted Fractures: Comminuted fracture of the vertebral body is characterized by disruption of the adjacent intervertebral disks and varying degrees of displacement of the fragments. The end plates of the body are forced into the centrum together with the disk. The extent of fragmentation (comminution) of the body depends upon the severity of the causative injury. A large fragment of the body may be displaced anteriorly; posteriorly displaced fragments are apt to cause compression of the cord or cauda equina; and the facet joints may be fractured or dislocated. Therefore, careful physical and x-ray examinations are mandatory before treatment is instituted.

The method of treatment may be dictated by neurologic findings. When cord or cauda equina injury has occurred, laminectomy and arthrodesis is the treatment of choice. When no neurologic involvement is present, reduction by hyperextension and immobilization in a plaster jacket is preferred. Bone healing is likely to be slow, and immobilization should be prolonged.

C. Fracture of the Transverse Processes: Fracture of the transverse processes may result from direct violence, such as a crushing injury, or may be incidental to a more serious fracture of the lumbar spine. It may result also from violent muscle contraction alone. One or more segments may be
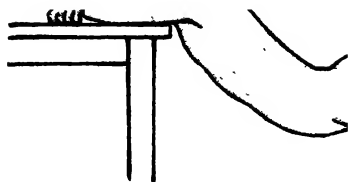
**Watson-Jones Technic of Reduction of Compression
Fracture of the Lumbar Spine**
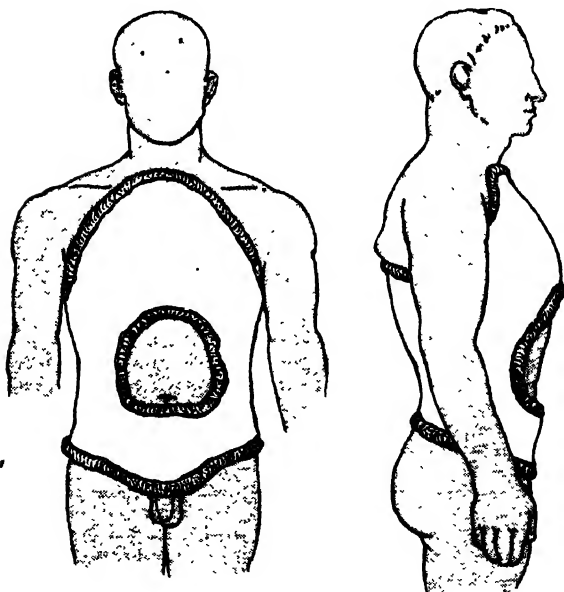
**Hyperextension Plaster for Compression
Fracture of the Lumbar Spine**

involved. If displacement is minimal, soft tissue injury is
likely to be minor. Extensive displacement of the fragments
indicates severe soft tissue tearing and hematoma formation.

Treatment depends upon the presence or absence of associ-
ated injuries. If fracture of the transverse process is the sole
injury, and if pain is not severe upon guarded motions of the
back, strapping and prompt ambulation may be sufficient. If
displacement and soft tissue injury are extensive, bed rest for
a few days followed by prolonged support in a corset or brace
may be necessary and slow recovery may be anticipated.

Fracture-Dislocation of the Lumbar Spine.

Severe compression trauma may cause fracture-dislocation
with rupture of 1 disk or, if comminution occurs, rupture of 2
disks. Varying degrees of injury to the posterior elements occur,
including unilateral or bilateral dislocation of the facets and/or
fracture of the pedicles or facets. Accompanying fractures of
spinous and transverse processes, and tearing of the posterior
ligaments and adjacent muscles can add to the complexity of this
severe lesion. The dislocation of the upper segment may be solely
in the anteroposterior plane, or it may be complex, with additional
displacement in the coronal plane with torsion around the longi-
tudinal axis of the spine.

Careful physical and radiologic examinations are necessary
to determine the nature of the injury before reduction is attempted,
since associated injury to the cord or cauda equina may be present.

The method of treatment depends upon the type of injury. If
there is no neurologic involvement and dislocation was associated
with fracture of the pedicles or facets, reduction may be attempted
by cautious extension under x-ray control without anesthesia. If
this is not successful, or if extension causes neurologic symptoms,
the attempt should be abandoned at once in favor of open reduction.

In complete dislocation of 1 or both facets, reduction can be
accomplished by open operation.

FRACTURE OF THE SACRUM

Fracture of the sacrum may accompany fracture of the pelvis.
It may also appear as an isolated lesion as a result of direct vio-
lence. Linear fracture of the sacrum without displacement should
be treated symptomatically. If the fracture extends through a
sacral foramen and is associated with displacement, there may be
injury to 1 of the sacral nerves and consequent neurologic deficit.
If the sacral fragment is displaced anteriorly, reduction should be
attempted by means of bimanual manipulation.

FRACTURE OF THE COCCYX

Fracture of the coccyx is usually the result of a blow on the
buttock. No specific treatment is required other than protection.
Strapping the buttocks together for a few days may minimize pain.
Pressure on the coccygeal region can be avoided by selecting a

firm chair in which to sit or placing a support beneath the thighs to relieve pressure. The patient should be warned that pain may persist for many weeks. Every effort toward conservative management should be made before coccygectomy is considered.

FRACTURES OF THE PELVIS

AVULSION FRACTURES OF THE PELVIS

Avulsion fractures of the pelvis include those involving the anterior-superior and anterior-inferior iliac spines and the apophysis of the ischium. The ischial apophysis may be avulsed indirectly by violent contraction of the hamstring muscles. If displacement is minimal, prompt healing without disability is to be expected. If displacement is marked (i. e., more than 1 cm.), reattachment by open operation is justifiable.

FRACTURE OF THE WING OF THE ILIUM

Isolated fracture of the wing of the ilium without involvement of the hip or sacroiliac joints most often occurs as a result of direct violence. With minor displacement of the free fragment, soft tissue injury is usually minimal and treatment is symptomatic. Wide displacement of the free fragment may cause extensive soft tissue injury with hematoma formation. Healing may occur by ossification of the hematoma with exuberant new bone formation.

ISOLATED FRACTURE OF THE OBTURATOR RING

Isolated fracture of the obturator ring, involving either the pubis or ischium with minimal displacement, is associated with little or no injury to the sacroiliac joints. This is also true of minor subluxation of the symphysis pubis. Initial treatment consists of bed rest for a few days followed by ambulation on crutches. A sacroiliac belt or pelvic binder may give additional comfort. As soon as discomfort disappears, unsupported weight-bearing may be permitted.

COMPLEX FRACTURES OF THE PELVIC RING

Complex fractures of the pelvic ring are due either to direct violence or to force transmitted indirectly through the lower extremities. They are characterized by disruption of the pelvic ring at 2 points: (1) anteriorly, near the symphysis pubis, manifested either by dislocation of that joint or by unilateral or bilateral fracture through the obturator ring; and (2) disruption of the pelvic ring through or in the vicinity of the sacroiliac joint. The disruption can extend partially through the sacroiliac joint as a dislocation and extend into the sacrum or into the adjacent ilium as a fracture.

The magnitude of displacement of the fragments may indicate the severity of soft tissue injury. These complex injuries are often associated with extensive hemorrhage into the soft tissues or injury to the bladder, urethra, or intra-abdominal organs. Anterior disruption may occur on 1 side and posterior disruption on the opposite side. The entire hemipelvis may be displaced proximally, or the pelvic ring may be widely opened.

When severe and complex fractures of the pelvic ring are suspected, the extent of associated injuries must be determined at once by physical and x-ray examination. Shock due to blood loss may be present.

Treatment is based upon the type and severity of the injury. If displacement and soft tissue injury are minimal, a pelvic sling to facilitate nursing care may be all that is required. When the hemipelvis has been displaced proximally, skeletal traction on the distal end of the femur on the affected side with suspension of the extremity may permit reduction.

If the sacroiliac joint has been dislocated and the ilium is rotated posterior to the sacrum, with opening of the anterior fracture, closed reduction can be attempted.

INJURIES OF THE SHOULDER GIRDLE

FRACTURE OF THE CLAVICLE

Fracture of the clavicle may occur as a result of direct violence or indirect violence transmitted through the shoulder. Most fractures of the clavicle are seen in the distal half, commonly at the junction of the middle and distal thirds. About two-thirds of clavicular fractures occur in children. Birth fractures of the clavicle vary from greenstick to complete displacement.

Because of the relative fixation of the medial fragment and the weight of the arm, the distal fragment is displaced downward and toward the midline. Anteroposterior x-rays should always be taken, but oblique projections are occasionally of more value. Injury to the brachial plexus or subclavian vessels is demonstrated on physical examination.

Treatment.*
  A. Without Displacement: Immobilization of "greenstick" fractures is not required, and healing is rapid. Complete fractures should be immobilized for 10-21 days. A figure-of-eight

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*Fracture of the outer third of the clavicle distal to the coracoclavicular ligaments is comparable to dislocation of the acromioclavicular joint. If the coracoclavicular ligaments are intact and the fragments are not widely displaced, immobilization in a sling and swathe is adequate. If the coracoclavicular ligaments have been lacerated and extensive displacement of the main medial fragment is present, treatment is similar to that advocated for acromioclavicular dislocation (see p. 545).

and carried obliquely upward over the acromial end of the clavicle, which has been padded with felt. An assistant then presses downward on the acromial end of the clavicle and lifts the patient's arm upward and backward. The strip of adhesive is then continued posteriorly along the upper arm and over the elbow, where another felt pad is placed over the olecranon and proximal ulna. The strip is then continued along the anterior aspect of the upper arm, across the acromial end of the clavicle, and posteriorly over the scapula. The hand and forearm are supported by a sling. This dressing must be maintained for at least 4 weeks; frequent adjustment is necessary to maintain immobilization in the correct position. The patient is encouraged to sleep in a semi-reclining position.

B. Complete Dislocation: It is difficult to maintain reduction and adequate immobilization of complete acromioclavicular dislocations by closed methods. Stimson's dressing or its modifications may be used successfully only if the patient can be kept under constant observation and frequent x-ray examinations made. Open reduction with temporary internal fixation is usually indicated until the ligaments have healed. Open reduction is most successful if it can be carried out within a few days after the injury. If treatment is deferred for 3 weeks or longer, the ligaments will have partially healed and become stretched, so that the deformity may be expected to recur when immobilization is discontinued.

STERNOCLAVICULAR DISLOCATION

Displacement of the sternal end of the clavicle may occur superiorly, anteriorly, or, less commonly, inferiorly. Retrosternal displacement is rare. Complete dislocation can be diagnosed by physical examination. Anteroposterior and oblique x-rays confirm the diagnosis.

Painful symptoms caused by post-traumatic degenerative arthritis from unreduced sternoclavicular dislocation may be disabling. Although repair of the sternoclavicular and costoclavicular ligaments may prevent further dislocation, pain may recur. Extraperiosteal resection of the medial two-thirds of the clavicle is indicated if symptoms persist.

For incomplete dislocation a plaster shoulder spica is adequate. Complete dislocations are not difficult to reduce, but external dressings are not adequate to maintain reduction; open reduction and temporary internal fixation of recent injuries are required. Immobilization for about 2 months is required.

FRACTURE OF THE SCAPULA

Unless fracture of the scapula is complicated by dislocation of the shoulder joint, no treatment is usually required except as noted below.

Fracture of the neck of the scapula is most often caused by a blow on the shoulder or by a fall on the outstretched arm. The degree of fragmentation varies from a crack to extensive commin-

ution. The main glenoid fragment may be impacted into the body fragment. The treatment of impacted or undisplaced fractures in patients 40 years of age or older should be directed toward the preservation of shoulder joint function, since stiffness may cause prolonged disability. In young adults especially, unstable fractures require arm traction with the arm at right angles to the trunk for about 4 weeks (see p. 550) and protection in a sling and swathe for an additional 2-4 weeks. Open reduction is rarely required even for major displaced fragments involving the glenoid.

Fracture of the acromion or spine of the scapula requires reduction only when the displaced fragment is apt to cause interference with abduction of the shoulder. Persistence of an acromial epiphysis should not be confused with fracture.

Fracture of the coracoid process may result from violent muscular contraction or may be associated with anterior dislocation of the shoulder joint.

Fracture of the body of the scapula may result from direct violence and may be complicated by fracture of underlying ribs. Treatment of uncomplicated fracture should be directed toward the comfort of the patient and the preservation of shoulder joint function.

FRACTURE OF THE PROXIMAL HUMERUS

Fracture of the Surgical Neck of the Humerus.

Fracture of the surgical neck is the most frequent fracture of the proximal humerus. It is encountered most often in patients in the older age group. It may occur as the result of a fall on the outstretched arm. Swelling of the shoulder region and restriction of motion due to pain are the prominent clinical features. The diagnosis is established by transthoracic and anteroposterior x-rays.

Undisplaced fractures require little treatment beyond the use of a sling and guarding of the shoulder until discomfort has disappeared. Restoration of bone continuity occurs in 8-12 weeks.

The head fragment may be impacted on the shaft in relative varus or valgus. An impacted fracture in marked varus may cause restriction of abduction, but the deformity is rarely sufficient to warrant disruption of the impaction and reduction. Impacted fractures of the surgical neck can be treated by means of a sling and swathe with early institution of active motion to preserve shoulder joint function.

Under anesthesia, attempted manipulation of unstable and displaced fractures of the surgical neck without complicating injury is justifiable, but in such instances the lesion must be stabilized by impacting or locking the fragments. When the shaft fragment has been displaced anteriorly and medially (in relation to the head fragment), prompt recurrence of the displacement may follow reduction if the fracture is not stable. Redisplacement is even more likely to occur with an unstable fracture which has been immobilized with the arm in abduction. Continuous traction, either by Buck's extension or by means of a Kirschner wire through the proximal ulna with the arm in abduction, and flexion of the elbow, is advisable when the fracture cannot be stabilized (see p. 550). This must be continued for about 4 weeks before partial healing causes stability.

An unstable fracture in varus position may be reduced by traction and abduction of the arm in external rotation, so that the shaft is brought into alignment with the head fragment. In this position the fracture becomes stable and can be immobilized in a spica. However, since in adults temporary stiffness of the shoulder is likely to follow if the shoulder is immobilized in abduction, the fracture should be stabilized with 1 or 2 stiff Kirschner wires (which can be introduced percutaneously). This will permit immobilization of the extremity at the side. The wires should be introduced obliquely in the deltoid region through the distal fragment into the head of the humerus. With the fragments stabilized, the arm is then brought to the side and immobilized either by a sling and swathe or by a plaster Velpeau dressing (see p. 552).

Fracture of the Greater Tuberosity of the Humerus.

Fracture of the greater tuberosity of the humerus with no associated injury is apt to be undisplaced, and may require little treatment other than relief of pain and preservation of shoulder joint function. Fracture of the greater tuberosity with displacement is likely to be associated with anterior dislocation of the shoulder joint.

Comminuted Fracture of the Proximal Humerus.

Comminuted fracture of the proximal humerus is not common, but its prognosis is unfavorable. The configuration of the fracture may vary, but 4 main fragments usually can be identified: 2 tuberosities, the head, and the shaft. Since little or no soft tissue remains, avascular necrosis of the head fragment is apt to occur. The head fragment may be completely displaced anterior or posterior to the glenoid, or it may be impacted on the shaft. In elderly patients, if the fracture is impacted and the head fragment is not dislocated, reduction should not be attempted. Early mobilization is indicated to preserve as much shoulder joint function as possible. If the head fragment is dislocated from the glenoid fossa, open reduction offers the best chance of salvaging shoulder joint function. If the blood supply to the head fragment has been destroyed, the fragment may be removed and the upper end of the humerus placed in the glenoid or a prosthesis inserted. The prognosis for functional recovery is poor in these cases.

Separation of the Epiphysis of the Head of the Humerus.

When this injury occurs as a result of birth trauma it is difficult to recognize because of the absence of a bony nucleus in the epiphysis. Even though x-ray examination is negative, the injury should be suspected when there is swelling of the shoulder region and limitation of motion of the arm. Fracture through the epiphysial plate may be encountered in older children. The principles of treatment are the same as for fracture of the surgical neck of the humerus. Open reduction is rarely desirable, and every effort should be made to obtain reduction by manipulation or traction.

DISLOCATION OF THE SHOULDER JOINT

Over 95% of all cases of shoulder joint dislocation are anterior or subcoracoid. Subglenoid and posterior dislocations comprise the remainder.

Anterior Dislocation of the Shoulder Joint.

Anterior dislocation presents the clinical appearance of prominence of the acromion, flattening of the deltoid region, anterior fullness, and restriction of motion due to pain. Both anteroposterior and axillary x-rays are necessary to determine the site of the head and the presence or absence of complicating fracture. Anterior dislocation may be complicated by (1) injury to 1 of the major nerves arising from the brachial plexus; (2) fracture of the upper extremity of the humerus, especially the head or greater tuberosity; and (3) tears of the capsulotendinous rotator cuff. The most common sequel is recurrent dislocation. Before manipulation, careful neurologic examination is necessary to determine the presence or absence of complicating nerve injury. Under general anesthesia, reduction can usually be accomplished by simple traction on the arm for a few minutes or until the head has been disengaged from the coracoid. If reduction cannot be obtained in this way, an assistant should apply lateral traction on the upper arm, close to the axilla, while the surgeon continues to exert traction on the arm. This is a modification of Hippocrates' manipulation in which the surgeon exerts traction on the arm with the heel of his unshod foot in the axilla for countertraction and forces the head of the humerus from beneath the acromion

If neither of the foregoing technics prove successful, Kocher's method may be useful. This maneuver, however, must be carried out gently or spiral fracture of the humerus may result. The elbow is flexed to a right angle and the surgeon applies traction and gentle external rotation to the forearm in the axis of the humerus. The elbow is then moved across the chest and the forearm rotated internally so that the palm rests on the opposite shoulder.

After closed reduction the extremity is immobilized in a sling and swathe for 3 weeks before active motion is begun.

Subcoracoid Dislocation of the Shoulder Joint.

Uncomplicated subcoracoid dislocation can almost always be reduced by closed manipulation. With associated fracture, or when the dislocation is old, open reduction may be necessary. Even when the dislocation is old, however, closed reduction by skeletal traction should be tried before open reduction is elected.

Posterior Dislocation of the Shoulder Joint.

Posterior dislocation is characterized by fullness beneath the spine of the scapula and by restriction of motion in external rotation. An axillary x-ray view demonstrates the position of the head of the humerus in relationship to the glenoid. This uncommon lesion may be reduced by the same combination of coaxial and transverse traction as described for anterior dislocation.

Recurrent Dislocation of the Shoulder Joint.

Recurrent dislocation of the shoulder is almost always anterior. Various factors can influence recurrent dislocation. Avulsion of
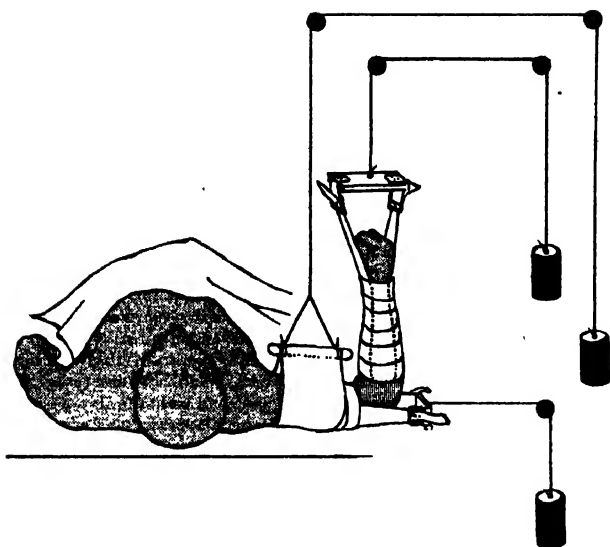
Skin Traction Applied to Upper Extremity
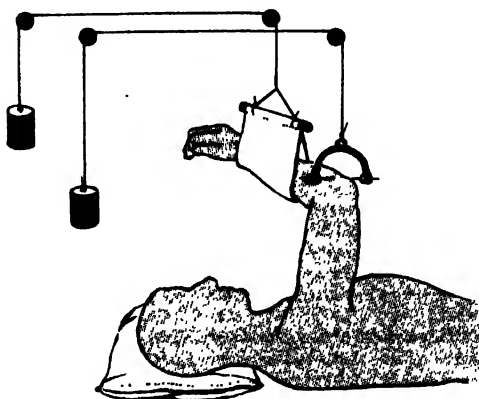
**Method of Suspension of Upper Extremity
With Skeletal Traction on Olecranon**

the anterior and inferior glenoid labrum or tears in the anterior
capsule remove the natural buttress that gives stability to the arm
with abduction and external rotation. Other lesions which impair
the stability of the shoulder joint are fractures of the posterior
and superior surface of the head of the humerus (or of the greater
tuberosity) and longitudinal tears of the rotator cuff between the
supraspinatus and subscapularis. Curative treatment must be
operative. After reduction the extremity is immobilized for 8
weeks in a Velpeau dressing or in a sling and swathe.

FRACTURES OF THE SHAFT OF THE HUMERUS

Fracture of the shaft of the humerus is more common in adults
than in children. Direct violence is accountable for the major por-
tion of such fractures, although spiral fracture of the middle third
of the shaft may result from violent muscular activity such as
throwing a ball. The diagnosis is based upon the history and phys-
ical examination. Localized tenderness, swelling, and deformity
are apparent. Palpation may elicit crepitus. Anteroposterior and
transthoracic x-rays show the location and configuration of the frac-
ture. Before initiating definitive treatment, a careful neurologic
examination should be done (and recorded) to determine the status
of the radial nerve. Injury to the brachial vessels is not common.
Fracture through the metaphysis proximal to the insertion of
the pectoralis major is classified as fracture of the surgical neck
of the humerus (see p. 547)

Fracture of the Upper Third of the Shaft of the Humerus.
Fractures between the insertions of the pectoralis major and
the deltoid commonly demonstrate adduction of the distal end of the
proximal fragment, with lateral and proximal displacement of the
distal fragment. Medial displacement occurs with fracture distal
to the insertion of the deltoid.
Treatment depends upon the presence or absence of compli-
cating neurovascular injury, the site and configuration of the
fracture, and the magnitude of displacement.
In infants, skin traction for 1-2 weeks will permit sufficient
callus to form so that immobilization can be maintained by a sling
and swathe or a Velpeau dressing. Open reduction for the sole
purpose of accurate positioning of the fragments is rarely justified
in children and adolescents, since slight shortening and minor de-
grees of angulation will be compensated during growth. Torsional
displacement, however, will not be compensated, and must be cor-
rected initially.
In the adult, an effort should be made to reduce completely
displaced transverse or slightly oblique fractures by manipulation.
An injection of 10-15 ml. of 2% procaine directly into the hematoma
at the fracture site will provide adequate anesthesia for manipula-
tion. If the ends of the fragments cannot be approximated by manip-
ulative methods, skin traction or skeletal traction is indicated with
a wire through the olecranon (see p. 550). In young patients the
olecranon wire should be placed opposite the coronoid process
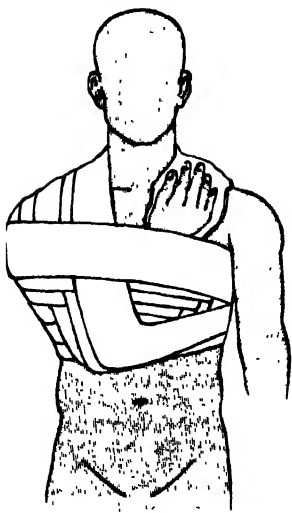
Velpeau Dressing
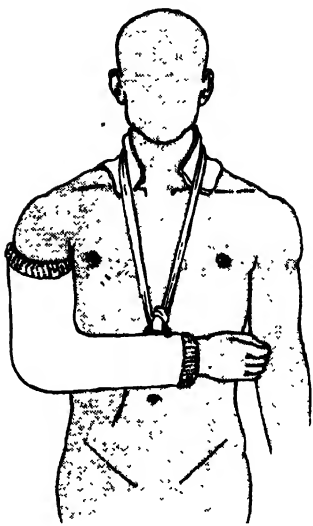
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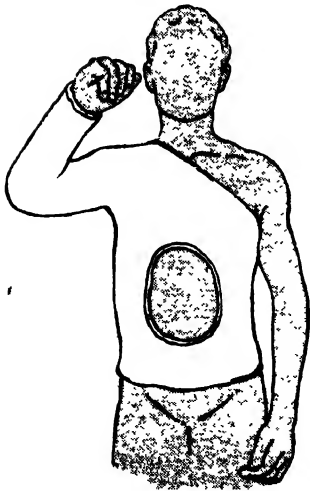
**Plaster Shoulder Spica
for Fracture of Humerus**

to avoid injury to the epiphysis. Traction should be continued for 3-4 weeks until stabilization occurs, after which time the patient can be ambulatory with an external immobilization device.

Fracture of the Middle and Lower Thirds of the Shaft of the Humerus.

Spiral, oblique, and comminuted fractures of the shaft below the insertion of the pectoralis major may be treated by Caldwell's hanging cast, which consists of a plaster dressing from the axilla to the wrist with the elbow in 90° of flexion and the forearm in mid-position (see p. 552). The cast is suspended from a bandage around the neck by means of a metal ring at the wrist. Alignment should be verified on anteroposterior and transthoracic x-rays with the patient standing. Angulation may be corrected by lengthening or shortening the suspension bandage. When lateral convex angulation cannot be corrected by adjustment of the bandage, moving the suspension ring closer to the elbow may be effective. Traction is afforded by the weight of the plaster. The patient is instructed to sleep in the semi-reclining position. As soon as clinical examination demonstrates stabilization (in about 6-8 weeks), the plaster may be discarded and a sling and swathe substituted.

If the configuration of the fracture approaches the transverse and is located between the insertions of the pectoralis major and the deltoid, the distal end of the proximal fragment may be in relative adduction. To prevent recurrence of medial convex angulation and maintain proper alignment, it may be necessary to bring the distal fragment into alignment with the proximal by bringing the arm across the chest and immobilizing it with a plaster Velpeau dressing (see p. 552).

When fracture of the shaft of the humerus is associated with other injuries which require confinement to bed, initial treatment may be by skin or skeletal traction (see p. 550). Continuous supervision is necessary.

Fractures of the shaft of the humerus - especially transverse fractures - may heal slowly. If stabilization has not taken place after 6-8 weeks of traction, more secure immobilization, such as with a plaster shoulder spica (see p. 552), must be considered. It may be necessary to continue immobilization for 6 months or more.

When complete loss of radial nerve function is apparent immediately after injury, open reduction is indicated to determine the type of lesion or to remove impinging bone fragments. If partial function of the radial nerve is retained, exploration can be deferred since spontaneous recovery sometimes occurs and may be complete by the time the fracture has healed. Open reduction of simple fractures is indicated also if arterial circulation has been interrupted or if adequate apposition of major fragments cannot be obtained by closed methods, as is likely to be the case with transverse fractures near the middle third of the shaft. When 4-5 months of treatment by closed methods have not resulted in clinical or x-ray evidence of healing, operative treatment may be considered.

INJURIES OF THE ELBOW REGION

FRACTURE OF THE DISTAL HUMERUS

Fracture of the distal humerus is most often caused by indirect violence. Therefore, the configuration of the fracture cleft and the direction of displacement of the fragments are likely to be typical. Soft tissue injuries and elbow joint dislocation are apt to be present.

Clinical findings consist of pain, swelling, and restriction of motion. Minor deformity may not be apparent because swelling usually obliterates landmarks. The type of fracture is determined by x-ray examination. Especially in children, it is advisable to obtain films of the opposite elbow for comparison.

Examination for peripheral nerve and vascular injury must be made and all findings carefully recorded before treatment is instituted.

Supracondylar Fracture of the Humerus.

Supracondylar fracture of the humerus occurs proximal to the olecranon fossa; transcondylar (diacondylar) fracture occurs distally and extends into the olecranon fossa. Neither fracture extends into the articular surface of the humerus. Treatment is the same for both types.

Since these fractures are observed more commonly in children and adolescents, they may extend into the epiphysial plates of the capitellum and trochlea.

The direction of displacement of the distal fragment from the midcoronal plane of the arm serves to differentiate the "extension" from the less common "flexion" type. This differentiation has important implications for treatment.

A. Extension Type Fractures: The significant direction of displacement of the distal fragment in the "extension" type of fracture is posterior and proximal. The distal fragment may also be displaced laterally and, less frequently, medially. The direction of these displacements is identified easily on biplane x-ray films. Internal torsional displacement, however, is more difficult to recognize; and unless torsional displacement is reduced, relative cubitus varus with loss of carrying angle will persist.

Displaced supracondylar fractures are surgical emergencies. Immediate treatment is required to limit circulatory embarrassment and peripheral nerve injury. If hemorrhage and edema prevent complete reduction of the fracture at the first attempt, a second manipulation will be required after swelling has regressed.

1. Manipulative reduction - Minor angular displacement (tilting) may be reduced by forced flexion of the elbow under local or general anesthesia, followed by immobilization in a posterior plaster splint in 45° of flexion (see p. 555). If displacement is marked but normal radial pulsation indicates that circulation is not impaired, closed manipulation under general anesthesia should be done as soon as possible. If radial pulses are absent or weak on initial examination
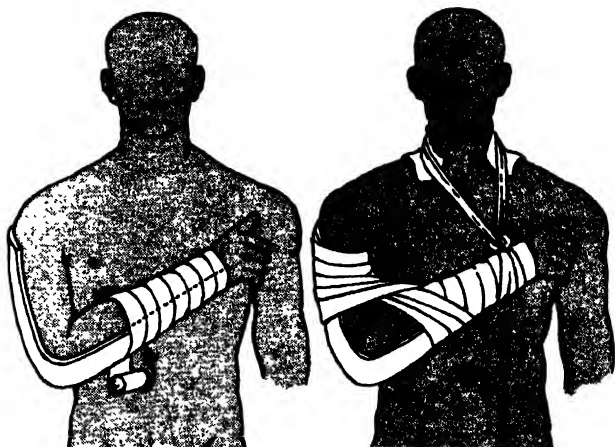
Posterior Plaster Splint for Supracondylar Fracture

and do not improve with manipulation, traction is indicated
(see below). After reduction and casting the patient should
be placed at bed rest, preferably in a hospital, with his el-
bow elevated on a pillow and the dressing arranged so that
the radial pulse is accessible for frequent observation.
Swelling can be expected to increase for 24-72 hours. Dur-
ing this critical period continued observation is necessary
so that any circulatory embarrassment which may lead to
Volkmann's ischemic contracture can be identified at once.
The circular bandage must be adjusted frequently to com-
pensate for the increase and decrease of swelling. If during
manipulation it was necessary to extend the elbow to less
than 45° to restore radial pulses, the joint may be flexed to
the optimal angle as swelling subsides.

In children stabilization will take place in 4-5 weeks,
after which time the plaster splint may be discarded and a
sling worn for another 2 weeks before active motion is per-
mitted. In adults healing is less rapid and immobilization
must be continued for 8-12 weeks or even longer before
active exercise is permitted.

Traction and immobilization - In certain instances supracon-
dylar fractures of the humerus with posterior displacement
of the distal fragment should be treated by traction (see p.
556): (1) If comminution is marked and stability cannot be
obtained by flexion of the elbow, traction is indicated until
the fragments have stabilized. (2) If 2 or 3 attempts at
manipulative reduction have been unsuccessful, continuous
traction under x-ray control for 1-2 days is justifiable be-
fore further manipulation. (3) If the radial pulse is absent
or weak when the patient is examined initially and does not
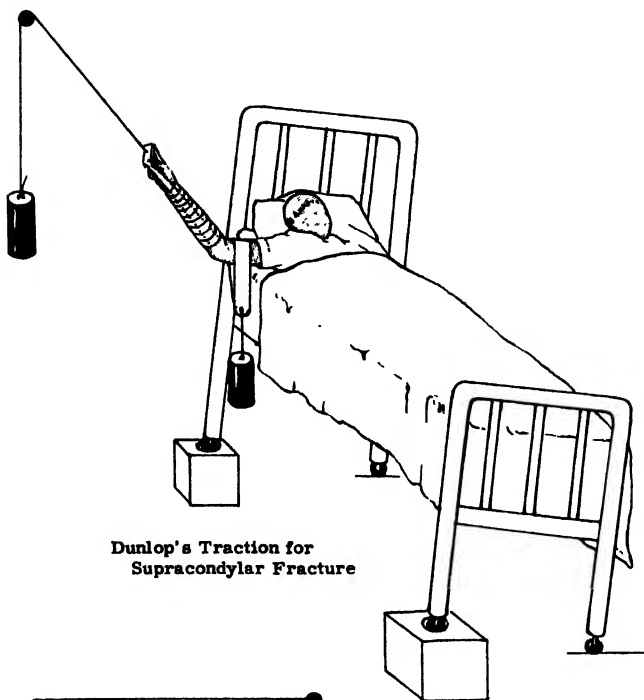
Dunlop's Traction for Supracondylar Fracture
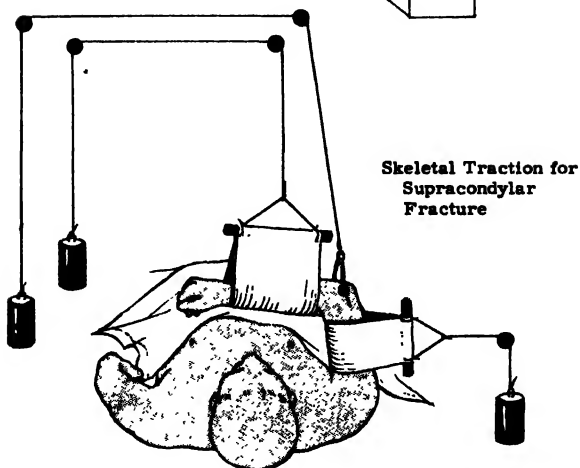
Skeletal Traction for Supracondylar Fracture

improve with manipulation, traction may be necessary to prevent displacement of the fracture. Acute flexion is contraindicated.

B. **Flexion Type Fracture:** Flexion type fracture of the humerus is characterized by anterior and sometimes also torsional and lateral displacement of the main distal fragment. Treatment is by closed manipulation. A posterior plaster splint is then applied from the axillary fold to the level of the wrist, with the forearm in supination and the elbow in full extension. Elevation is advisable for 24 hours or until soft tissue swelling is under control, after which time the patient may be ambulatory. Immobilization is then continued for 4-6 weeks.

Separation of the Distal Humeral Epiphyses.

An uncommon variation of supracondylar fracture is separation of the distal humeral epiphyses with or without appreciable displacement. Sprains of the elbow do not commonly occur in children; injury more often involves the distal humeral epiphyses. X-ray comparison of the injured elbow with the uninjured elbow may show no deviation, but careful physical examination may demonstrate posterior tenderness over the lower epiphyses in addition to swelling. This combination of swelling and tenderness should suggest separation of the epiphyses, and warrants protection from further injury by means of a sling worn for about 3 weeks.

The direction of displacement is determined by careful clinical and radiographic examinations. Depending upon the direction of angulation of the osseous nuclei of the capitellum and trochlea (as demonstrated in the lateral x-ray), immobilization is as described for supracondylar fractures.

Intercondylar Fracture of the Humerus.

Intercondylar fracture of the humerus is classically described as being of the "T" and/or "Y" type, according to the configuration of the fracture cleft observed on an anteroposterior x-ray. This fracture is usually seen in adults, commonly as the result of a blow over the posterior aspect of the flexed elbow. Open fracture and other injuries to the soft tissues are frequently present. The fracture often extends into the trochlear surface of the elbow joint, and unless the articular surfaces of the distal humerus can be accurately repositioned, restriction of joint motion, pain, instability, and deformity can be expected.

A. **Closed Reduction:** If the fragments are not widely displaced, closed reduction may be successful. Since comminution is always present, stabilization is difficult to achieve and maintain by manipulation and external immobilization.

1. Anterior displacement may be treated first by a combination of continuous skin traction with the elbow in full extension and closed manipulation of the main fragments. If adequate positioning can be achieved in this manner, traction is continued until stabilization occurs. The extremity may then be immobilized in a tubular plaster cast.

2. Posterior displacement requires overhead skeletal traction by means of a Kirschner wire inserted through the olecranon (see p. 556). It may be necessary to apply a swathe around the arm and body for simultaneous transverse traction.

B. Open reduction is indicated if adequate positioning cannot be obtained by closed methods.

Fracture of the Lateral Condyle of the Humerus.

The 3 major varieties of fracture of the lateral condyle of the humerus are (1) fracture of a portion of the capitellum in the coronal plane of the humerus, with or without extension into the trochlea (seen only in adults); (2) isolated fracture of the lateral condyle without extension into the trochlea; and (3) separation of the capitellar epiphysis (in children).

A. Fracture of the Capitellum: This is characterized by proximal displacement of the anterior detached fragment, and probably occurs as 1 component of a spontaneously reduced incomplete dislocation of the elbow joint. The essential lesion is most clearly demonstrated on lateral x-rays. Closed reduction should be attempted. After reduction the extremity is immobilized in a posterior plaster splint with the elbow in full flexion.

B. Isolated complete fracture of the lateral condyle without extension into the trochlea*is uncommon, and is not usually associated with major displacement of the detached fragment. The extremity should be supported by a sling. If tense hemarthrosis is present, aspiration may minimize pain. Guarded active motions of the elbow should be initiated as soon as pain subsides.

C. Fracture of the lateral condyle of the humerus in children is essentially separation of the capitellar epiphysis, even though the fracture may extend into the metaphysis and the trochlear epiphysis. If the center of ossification of the capitellum is small, minor displacement may be missed on initial examination; further displacement will then result from unguarded use. The fact that the extensor muscles originate on the fragment is an important factor in displacement.

1. Closed reduction - Minor displacement may be treated by manipulative reduction and external immobilization in a posterior plaster splint which extends from the posterior axillary fold to the level of the heads of the metacarpals. X-rays are taken at least twice a week for the first 3 weeks to determine whether displacement has recurred.

2. Open reduction - When anatomic reduction cannot be achieved by 1 or 2 manipulations, open reduction is indicated.

Avulsion of the Medial Epicondylar Apophysis.

Avulsion of the medial epicondylar apophysis in children may occur without dislocation of the elbow. Minor displacement causing localized tenderness and swelling over the medial aspect of the elbow can be treated by immobilization in a sling and swathe for a few days. More extensive injury should be suspected if tenderness and swelling are diffuse. When separation is greater than 1-2 mm., treatment is similar to that for dislocation of the elbow associated with separation of the apophysis (see p. 561).

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*Fracture which involves the entire capitellum and extends into the trochlea is associated with proximal and lateral displacement of the detached fragment and lateral subluxation of the elbow. This lesion is discussed on p. 562.

FRACTURE OF THE PROXIMAL ULNA

Common fractures of the proximal ulna include fracture of the olecranon and fracture of the coronoid process. Fracture of the coronoid process is a complication of posterior dislocation of the elbow joint, and is discussed on p. 561.

Fracture of the olecranon which occurs as the result of indirect violence (e.g., forced flexion of the forearm against the actively contracted triceps muscle) is typically transverse or slightly oblique. Fracture due to direct violence is usually comminuted and associated with other fracture or anterior dislocation of the joint. Since the major fracture cleft extends into the elbow joint, treatment should be directed toward restoration of anatomic position to afford maximal recovery of range of motion and muscular activity.

Treatment.

The method of treatment depends upon the degree of displacement and the extent of comminution.

A. Closed Reduction: Minimal displacement (1-2 mm.) can be treated by closed manipulation with the elbow in full extension, assisted by digital pressure over the proximal fragment, and immobilization in a volar plaster splint which extends from the anterior axillary fold to the wrist. X-rays should be taken twice weekly for 2 weeks after reduction to determine whether reduction has been maintained. Immobilization must be continued for at least 6 weeks before active flexion exercises are begun.

B. Open reduction and internal fixation are indicated if closed methods are not successful. The extremity is then immobilized in 90° of flexion for at least 6-8 weeks before active flexion exercises are instituted.

FRACTURE OF THE PROXIMAL RADIUS

Fracture of the Head and Neck of the Radius.

Fracture of the head and neck of the radius may occur in adults as an isolated injury uncomplicated by dislocation of the elbow or the superior radioulnar joint. This fracture is caused by indirect violence, such as a fall on the outstretched hand, when the radial head is driven against the capitellum. Care must be taken to obtain true anteroposterior and lateral x-rays of the proximal radius as well as of the elbow joint, since minor lesions may be obscured by a change in position from midposition to full supination during exposure of the films.

A. Conservative Measures: Fissure fractures and those with minimal displacement can be treated symptomatically, with evacuation of tense hemarthrosis by aspiration to minimize pain. The extremity may be supported by a sling or immobilized in a posterior plaster splint with the elbow in 90° of flexion. Active exercises of the elbow are to be encouraged within a few days. Recovery of function is slow, and slight restriction of motion (especially extension) may persist.

B. Surgical Treatment: When the fracture is comminuted, or when displacement is greater than 1-2 mm., excision of the entire head of the radius is desirable.

Fracture of the Upper Epiphysis of the Radius.

Fracture of the upper radial epiphysis in a child is not a true epiphysial separation since the fracture cleft commonly extends into the neck of the bone. Because the articular surface of the proximal fragment remains intact, the prominent features of displacement are angulation and impaction. Wide displacement of the minor fragment may mean that the elbow joint was dislocated but has reduced spontaneously since the injury.

A. Closed Reduction: Every effort should be made to reduce these fractures by closed manipulation. Several x-rays taken with the forearm in various degrees of rotation should be examined so that the position can be selected which is best suited for digital pressure on the proximal fragment. Anteroposterior and lateral x-rays with the elbow in flexion are then taken; if angulation has been reduced to less than 45°, the end result is considered satisfactory.

B. Open Reduction: If closed reduction is not successful, open reduction and repositioning under direct vision is indicated.

SUBLUXATION AND DISLOCATION OF THE ELBOW JOINT

Subluxation of the Head of the Radius.

This injury occurs most frequently in infants between the ages of 18 months and 3 years, usually when the child is suddenly lifted by his hand. In unreduced subluxations, swelling and tenderness are present in the region of the ulnar head at the level of the inferior radioulnar joint and the infant holds his forearm semiflexed and pronated. If spontaneous reduction has occurred, the diagnosis is dependent upon the finding of restricted supination associated with discomfort. X-rays are not helpful.

Reduction by forced supination of the forearm can be accomplished easily without anesthesia. The arm should be protected in a sling for 1 week to prevent recurrence.

Dislocation of the Elbow Joint Without Fracture.

Dislocation of the elbow joint without major fracture is almost always posterior. It may be encountered at any age, but is most common in children. Complete backward dislocation of the ulna and radius implies extensive tearing of the capsuloligamentous structures and injury to the region of insertion of the brachialis muscle. The coronoid process of the ulna is usually displaced posteriorly and proximally into the olecranon fossa, but it may be displaced laterally or medially. Biplane x-rays of the highest quality are necessary to determine that no fracture is associated.

Peripheral nerve function must be carefully assessed before definitive treatment is instituted. The ulnar nerve is most likely to be injured.

In recent dislocations, closed reduction can be achieved (under general anesthesia) by axial traction on the forearm with the elbow in the position of deformity. Hyperextension is not necessary. Lateral or medial dislocation can be corrected during traction. As soon as proximal displacement is corrected, the elbow should be brought into 90° of flexion and a posterior plaster splint applied

which reaches from the posterior axillary fold to the wrist. Active motion is permitted after 3 weeks.

Closed reduction should be attempted even if unreduced dislocation has persisted for 2 months following the injury.

FRACTURE-DISLOCATION OF THE ELBOW JOINT

Dislocation of the elbow is frequently associated with fracture. Some fractures are insignificant and require no specific treatment; others demand specialized care.

Fracture of the Coronoid Process of the Ulna.

Fracture of the coronoid process of the ulna is the most frequent complication of posterior dislocation of the elbow joint. Treatment is the same as for uncomplicated posterior dislocation of the elbow joint.

Fracture of the Head of the Radius With Posterior Dislocation of the Elbow Joint.

This injury is treated as 2 separate lesions. The severity of comminution and the magnitude of displacement are first determined by x-ray. If comminution has occurred or the fragments are widely displaced, the dislocation is reduced by closed manipulation and the head of the radius is then excised.

If fracture of the head of the radius is not comminuted and the fragments are not widely displaced, treatment is as for uncomplicated posterior dislocation of the elbow joint (see above).

Fracture of the Olecranon With Anterior Dislocation of the Elbow Joint.

This very unstable injury usually occurs from a blow on the dorsum of the flexed forearm. Fracture through the olecranon permits the distal fragment of the ulna and the proximal radius to be displaced anterior to the humerus, and may cause extensive tearing of the capsuloligamentous structures around the elbow joint. The dislocation can be reduced by bringing the elbow into full extension, but anatomic reduction of the olecranon fracture by closed manipulation is not likely to be successful and immediate open reduction is usually indicated. Recovery of function is likely to be delayed and incomplete.

Fracture of the Medial Epicondylar Apophysis With Dislocation of the Elbow Joint.

Dislocation of the elbow joint in children may be complicated by avulsion of the medial epicondylar apophysis. The direction of dislocation may have been lateral, posterior, or posterolateral. Physical and x-ray examination may not demonstrate the extent of displacement at the time of injury since partial reduction may have occurred spontaneously. X-rays of the uninjured elbow in similar projections are desirable to compare the exact locations of the 2 apophyses. The free fragment is displaced downward by the flexor muscles. If partial spontaneous reduction has occurred, the detached apophysis may be found incarcerated within the elbow joint between the articular surfaces of the trochlea and the olecranon.

This may happen also during manual reduction. Ulnar nerve function must be evaluated before definitive treatment is given.

Dislocation of the elbow joint may be reduced by closed manipulation, but accurate repositioning of a widely separated apophysis cannot be achieved by closed methods. Opinion differs concerning the necessity for anatomic reduction of the apophysis if it is not incarcerated within the elbow joint. Some authorities maintain that fibrous healing of the apophysis causes no disability; others anticipate weakness of grasp. Exuberant bone formation around the apophysis may cause tardy ulnar paralysis. If it is elected not to reduce displacement of an apophysis outside the elbow joint, the extremity should be immobilized at a right angle for 3 weeks in a tubular plaster cast before active motion is permitted.

If the ulnar nerve has been injured, or if the apophysis cannot be displaced from the elbow joint by closed manipulation, open reduction is advisable.

Fracture of the Lateral Condyle With Lateral Dislocation of the Elbow Joint.

Fracture of the lateral condyle of the humerus with lateral dislocation of the elbow joint must be differentiated from fracture of the lateral condyle with or without posterior dislocation of the joint (see below). Neither lesion is common. A complicating feature of fracture of the lateral condyle with lateral dislocation is inclusion not only of the entire capitellum but also extension of the fracture cleft into the trochlea. This creates an unstable mechanism which cannot be reliably immobilized in either flexion or extension even though closed reduction has been successful. If closed methods of treatment are not adequate, open reduction is recommended.

Fracture of the Lateral Condyle With Posterior Dislocation of the Elbow Joint.

The dislocation should be treated before the fracture is reduced.

INJURIES OF THE SHAFTS OF THE RADIUS AND ULNA

FRACTURES OF THE SHAFTS OF THE RADIUS AND ULNA

General Considerations.
  A. Causative Injury: Spiral and oblique fractures are likely to be caused by indirect injury. Greenstick, transverse, and comminuted fractures are commonly the result of direct injury.
  B. Radiography:
    1. In addition to anteroposterior and lateral films of the entire forearm, including the elbow and wrist joints, oblique views are often desirable.
    2. The lateral projection is usually taken with the forearm in midposition (between complete pronation and supination).

3. For the anteroposterior projection care must be taken to prevent any change in relative supination of the radius; if this happens, the distal radius will be the same in both views.

4. Especially in children, films of the uninjured forearm are desirable for comparison of epiphyses and for future reference if growth is impaired.

C. Anatomic Peculiarities: Both the radius and the ulna have biplane curves which permit 180° of rotation in the forearm. If the curves are not preserved by reduction, full motion of the forearm may not be recovered or derangement of the radioulnar joints may follow.

Torsional displacement by muscle activity has important implications for manipulative treatment of certain fractures of the radial shaft. The direction of torsional displacement of the distal fragment following fracture of the shaft is influenced by the location of the lesion in reference to muscle insertion. If the fracture is in the upper third (above the insertion of the pronator teres), the proximal fragment will be drawn into relative supination by the biceps and supinator and the distal fragment into pronation by the pronator teres and pronator quadratus. The relative position in torsion of the proximal fragment may be determined by comparing the position of the bicipital tubercle on an anteroposterior film with similar projections of the uninjured arm taken in varying degrees of forearm rotation. In fractures below the middle of the radius (below the insertion of the pronator teres), the proximal fragment characteristically remains in midposition and the distal fragment is pronated; this is due to the neutralizing action of the pronator teres on the biceps and supinator.

D. Closed Reduction and Splinting: With fracture of the shaft of either the radius or the ulna, with displacement, injury of the proximal or distal radioulnar joints should always be suspected. The presence of swelling and tenderness around the joint may aid in localization of an occult injury when x-rays are not helpful.

In both adults and children, closed reduction of uncomplicated fractures of the radius and ulna should be attempted. The type of manipulative maneuver depends upon the configuration and location of the fracture and the age of the patient. The position of immobilization of the elbow, forearm, and wrist depends upon the location of the fracture and its inherent stability.

Fracture of the Shaft of the Ulna.

Isolated fracture of the shaft of the proximal third of the ulna (above the insertion of the pronator teres) with displacement is often associated with dislocation of the head of the radius. Reduction of an undislocated transverse fracture may be achieved by axial traction followed by digital pressure to correct displacement in the transverse plane. With the patient supine, the hand is suspended overhead and counter-traction is provided by a sling around the arm above the flexed elbow. After the fragments are distracted, transverse displacement is corrected by digital pressure. With the elbow at a right angle and the forearm in midposition, the extremity is then immobilized in a tubular plaster cast extending from the

axilla to the metacarpophalangeal joints. During the first month, weekly examination by x-ray is necessary to make certain that displacement has occurred. Immobilization must be maintained until bone continuity is restored (usually in 8-12 weeks).

Fracture of the shaft of the ulna distal to the insertion of the pronator teres is apt to be complicated by angulation. The proximal end of the distal fragment is displaced toward the radius by the pronator quadratus muscle. Reduction can be achieved by the maneuver described above. To prevent recurrent displacement of the distal fragment, the plaster cast must be carefully molded so as to force the mass of the forearm musculature between the radius and ulna into the anteroposterior plane. Care should be taken to avoid pressure over the subcutaneous surfaces of the radius and ulna around the wrist. Healing is slow, and frequent radiologic examination is necessary to make certain that displacement has not occurred. Stabilization may require 4 months of immobilization.

An oblique fracture cleft creates an unstable mechanism with a tendency toward displacement. Open reduction and rigid internal fixation with bone plates or an intramedullary rod are indicated.

Open reduction of uncomplicated fracture of the ulna in children is rarely justifiable because accurate reduction is not imperative; in children under 12 years of age an angular deformity as great as 15° may be corrected by growth. Torsional displacement of uncomplicated fractures of the shaft is not likely to occur. Deformity caused by transverse displacement will be corrected by growth and remodeling.

Fracture of the Shaft of the Radius.

Isolated closed fracture of the shaft of the radius can be caused by direct or indirect violence; open fracture usually results from penetrating injury. Closed fracture with displacement is usually associated with other injury (e.g., fracture of the ulna or dislocation of the distal radioulnar joint). X-rays may not reveal dislocation, but localized tenderness and swelling suggest injury to the distal radioulnar joint.

If the fracture is proximal to the insertion of the pronator teres, closed reduction is indicated. The extremity should then be immobilized in a tubular plaster cast which extends from the axilla to the metacarpophalangeal joints, with the elbow at a right angle and the forearm in full supination (see below).
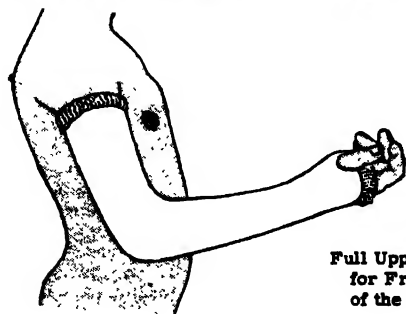
Full Upper Extremity Plaster for Fracture of Both Bones of the Forearm

If the fracture is distal to the insertion of the pronator teres, manipulation and immobilization are as described above except that the forearm should be in midrotation rather than full supination. Since injury to the distal radioulnar joint is apt to be associated with fracture of the radial shaft below the insertion of the pronator teres, weekly anteroposterior and lateral x-ray projections should be taken during the first month to determine the exact status of reduction.

If the configuration of the fracture cleft is transverse rather than oblique, displacement is less apt to take place following anatomic reduction. In the adult, if stability cannot be achieved or if reduction does not approach the anatomic, open reduction and internal fixation are recommended since deformity as a result of displacement of fragments is likely to cause limitation of forearm and hand movements. Children under 12 years of age are likely to recover function provided that torsional displacement has been corrected and angulation does not exceed 15°. Especially if it is convex anteriorly, angulation greater than 15° should be corrected in children even though open reduction is required.

In adults, a snug plaster should be maintained for 8-12 weeks or even longer, since healing may be slow. Healing is rapid in children even though reduction is not anatomic; open reduction to promote bone healing in children is not necessary.

Fracture of the Shafts of Both Bones.

The management of fractures of the shafts of both bones of the forearm is essentially a combination of those technics which have been described for the individual bones. If both bones are fractured at the same time, dislocation of either radioulnar joint is not likely to occur. If the configuration of the fracture cleft is approximately transverse, stability can be attained by closed methods provided reduction is anatomic or nearly so. Oblique or comminuted fractures are unstable.

Treatment depends in part upon the degree of displacement, the severity of comminution, and the age of the patient.

A. Without Displacement: In adults, fracture of the shaft of the radius and ulna without displacement can be treated by immobilization in a tubular plaster cast extending from the axilla to the metacarpophalangeal joints with the elbow at a right angle and the forearm in supination (fractures of the upper third) or midposition (fractures of the mid and lower thirds). Immobilization is maintained for 8-12 weeks or until bone continuity is restored. To avoid late angulation or refracture, the elbow should be included in the plaster until the callus is well mineralized.

B. Greenstick Fractures: Greenstick fractures of both bones of the forearm are common in children.

1. With fractures of the lower third in children under 12 years of age, if angulation is no more than 15° and convex posteriorly, satisfactory correction of the deformity can be expected to occur spontaneously with growth. If angulation is greater than 15° or if the apex is directed anteriorly, deformity should be corrected and the extremity immobilized in a tubular plaster cast extending from the axilla to the bases of the fingers, the elbow at a right angle, the forearm

      in pronation, and the wrist in the neutral position. Reduction is maintained by snug anteroposterior molding of the plaster over the distal third of the forearm rather than by placing the wrist in volar flexion.

2. Greenstick fracture of both bones proximal to the distal third of the shaft has a tendency toward increased angular deformity if angulation alone is corrected without completion of the fracture. It is recommended that the fracture be completed by sharply reversing the direction of angulation until a palpable "snap" indicates that intact fibers of bone and periosteum on the convex surface have ruptured. The extremity is then immobilized in a plaster cast similar to that used for lower third fractures, with the forearm in semisupination.

C. With Displacement: Although it is not always possible to correct displaced fractures of both bones of the forearm by closed methods, an attempt should be made to do so both in adults and in children if x-ray studies show a configuration which can be stabilized without operation. Manipulative reduction is recommended if the patient is seen soon after injury and overriding is less than 1 cm. It is essential that good apposition of the fragments of each bone be obtained. Once adequate reduction has been achieved, and while traction is maintained, a padded tubular plaster cast is applied from the bases of the fingers to the axilla.
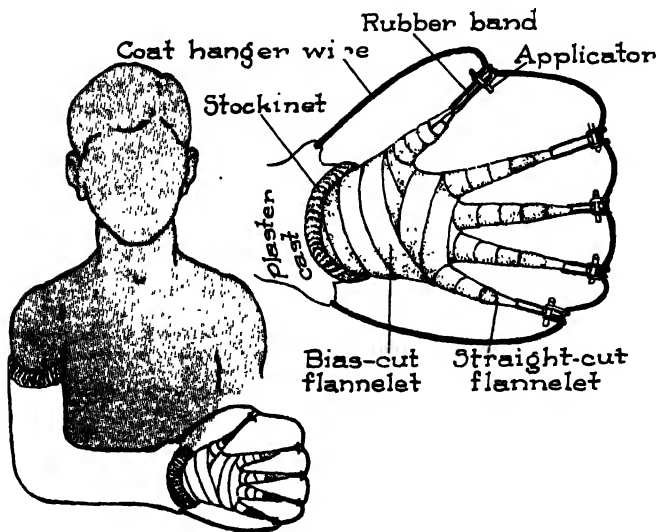
**Banjo Splint and Skin Traction for Fracture
of the Forearm in Children (Blount)**

If treatment is delayed until hemorrhage and swelling have caused induration by infiltration of the soft tissues, or if overriding is more than 1 cm., sustained traction for 2-3 hours will probably be necessary to overcome shortening. Skin traction and soft tissue countertraction are hazardous in these circumstances because of the possibility of decubiti or vascular injury. Skeletal traction is indicated. When correction of the overriding is demonstrated by x-ray, the fragments are manipulated into position under local or general anesthesia. A plaster cast with wires incorporated is then applied and the bows maintained to keep the wires taut.

Persistent overriding without angulation in children is not a problem, since 0.5 cm. of shortening may be corrected by growth. If overriding of more than 0.5 cm. is demonstrated, continuous skin traction upon the fingers with elastic bands attached to a banjo loop incorporated into the tubular plaster is indicated (Blount's technic) (see p. 566).

In adults, if accurate apposition of fragments or stability cannot be achieved in fractures of both bones, open reduction and internal fixation are recommended provided that experienced personnel and adequate equipment are available. Persistent displacement of the fragments of 1 or both bones may cause delay of healing (due to soft tissue interposition), restriction of forearm movements, derangement of the radioulnar joints, and deformity. In those fractures in which open reduction is justifiable in the adult, rigid internal fixation is indicated; a technical pitfall to be avoided is the use of a single wire loop or transfixation screw, a short bone plate attached with unicortical screws, or small intramedullary wires. Even though excellent stability is achieved at operation with internal devices, the extremity should be protected by external fixation until bone healing is well under way.

FRACTURE-DISLOCATIONS OF THE RADIUS AND ULNA

Fracture of the Ulna With Dislocation of the Radial Head.
Fracture of the shaft of the ulna near the junction of the middle and upper thirds may be associated with anterior or posterior dislocation of the head of the radius (Monteggia's fracture). Lateral dislocation of the head of the radius with convex angulation of the shaft of the ulna in the same direction is not common.

A. Closed Reduction:

1. Anterior dislocation of the head of the radius - Although this lesion is usually caused by direct violence upon the dorsum of the forearm, it may also be caused by forced pronation. The annular ligament may be torn, or the head may be displaced distally from beneath the annular ligament without causing a significant tear. The injured ligament may be interposed between the articular surface of the head of the radius and the capitellum or ulna.

Closed reduction can be achieved under general anesthesia. Lateral and posteroanterior x-rays are then taken, and a posterior plaster splint is applied from the axillary

fold to the head of the metacarpals with the elbow in 45° of flexion (see p. 555) while the films are prepared. If reduction is anatomic, the arm is elevated and observed frequently for signs of circulatory embarrassment for at least 72 hours. Bandages must be adjusted at appropriate intervals to prevent displacement of the splint. As soon as the dressings have been applied, the x-ray examination should be repeated. Examination should be done again on the third day after reduction. Thereafter, for at least 1 month, biplane x-rays are taken once a week. Immobilization is maintained until bone continuity of the ulna is restored; this usually requires 10-12 weeks or even longer, since healing is likely to be slow. Acute flexion of the elbow in plaster should not be decreased before the eighth week.

2. Posterior dislocation of head of the radius - This lesion is caused by direct violence to the volar surface of the forearm. Treatment is by closed reduction. Anteroposterior and lateral x-rays are then taken, and a tubular plaster cast or stout posterior plaster splint is applied from the metacarpal heads to the axilla with the elbow in full extension and the forearm in midposition. Careful postreduction observation as for anterior dislocation is essential.

B. Open Reduction: If accurate reduction of the fracture and the dislocation cannot be achieved by closed methods, open reduction by the technic of Boyd is indicated. Plaster immobilization is indicated until healing is well under way.

Fracture of the Shaft of the Radius With Dislocation of the Ulnar Head.

In fracture of the shaft of the radius near the junction of the middle and lower thirds with dislocation of the head of the ulna (Dupuytren's fracture, Galeazzi's fracture), the apex of major angulation is usually directed anteriorly while the ulnar head lies volar to the distal end of the radius. (Convex dorsal angulation with the ulnar head posterior to the lower end of the radius is rare.)

A. Closed Reduction: Anatomic alignment is difficult to obtain by closed manipulation and difficult to maintain in plaster, but these technics should be tried before open reduction is used. After reduction, anteroposterior and lateral x-rays are taken and developed before application of the cast. If reduction is adequate, a tubular plaster cast is applied from the axilla to the knuckles with the elbow at a right angle, the forearm in pronation, and the wrist in neutral position. Weekly x-ray examination is indicated during the first month. Immobilization in a snug tubular plaster cast is continued until healing of the radius is complete.

Immobilization of the rare posterior type is with the forearm in supination.

B. Open Reduction: If anatomic reduction cannot be achieved by closed methods, open reduction of recent fracture of the radius is recommended.

INJURIES OF THE WRIST REGION

SPRAINS OF THE WRIST

Sprain of the ligaments of the wrist joint is not common, and the diagnosis of wrist sprain should not be made until other lesions, e.g., injury to the lower radial epiphysis (in children) and carpal fractures and dislocations (in adults) have been ruled out. If symptoms persist for more than 2 weeks, and especially if pain and swelling are present, x-ray examination should be repeated.

Treatment may be by immobilization with a volar splint extending from the palmar flexion crease to the elbow. The splint should be attached with elastic bandages so that it can be removed at least 3 times daily for gentle active exercise and warm soaks.

COLLES' FRACTURE

Abraham Colles described the fracture that bears his name as an impacted fracture of the radius 1.5 inches above the wrist joint. Modern usage has extended the term "Colles' fracture" to include a variety of complete fractures of the distal radius characterized by dorsal displacement of the distal fragment.

The fracture is commonly caused by a fall with the hand outstretched, the wrist in dorsiflexion, and the forearm in pronation, so that the force is applied to the palmar surface of the hand. Colles' fracture is most common in middle life and old age.

The fracture cleft may be transverse or oblique, and extends across the distal radius. It may be comminuted, extending into the radiocarpal joint. Displacement is often minimal, with dorsal impaction caused by tilting of the distal fragment and volar convex angulation. As displacement becomes more marked, dorsal and radial tilt of the distal fragment causes increased angulation and torsional displacement in supination of the distal fragment. The normal volar and ulnar inclination of the carpal articular surface of the radius is reduced or reversed.

Avulsion of the styloid process is the usual injury to the distal ulna. Extension of the fracture cleft into the ulnar notch may injure the distal radioulnar articulation. The carpus is displaced with the distal fragment of the radius. Marked displacement at the fracture site causes dislocation of the distal radioulnar and ulnocarpal articulations, and tearing of the triangular fibrocartilage, both radioulnar ligaments, and the volar ulnocarpal ligaments. If the ulnar styloid is not fractured, then the collateral ulnar ligament is torn. The head of the ulna lies anterior to the distal fragment of the radius.

Clinical Findings.

Clinical findings vary according to the magnitude of injury, the degree of displacement of fragments, and the interval since injury. If the fragments are not displaced, examination soon after injury will demonstrate only slight tenderness and insignificant swelling; pain may be absent. Marked displacement produces the classical

"silver fork" or "bayonet" deformity, in which a dorsal prominence caused by displacement of the distal fragment replaces the normal convex curve of the lower radius and the ulnar head is prominent on the anteromedial aspect of the wrist. Later, swelling may extend from the fingertips to the elbow.

Complications.

Derangement of the distal radioulnar joint is the most common complicating injury. Median nerve injury is usually mild and recovery prompt (within 6 months). Other complicating injuries are fracture of the carpal navicular, the head of the radius, or the capitellum. Dislocation of the elbow and shoulder and tears of the capsulotendinous cuff of the shoulder may be associated.

Treatment.

Complete recovery of function and a good cosmetic result are goals of treatment which cannot always be achieved. The patient's age, sex, and occupation, the presence of complicating injury or disease, the severity of comminution, and the configuration of the fracture cleft govern the selection of treatment.

Open reduction of recent closed Colles' fracture is rarely the treatment of choice. Many technics of closed reduction and external immobilization have been advocated; the experience and preference of the surgeon determine the selection.

A. Minor Displacement: Colles' fracture with minimal displacement is characterized by absence of comminution and slight dorsal impaction. Deformity is barely perceptible, or may not be visible even to a trained observer. In the elderly patient, treatment is directed toward early recovery of function. In young patients, prevention of further displacement is the first consideration.

Reduction is not necessary. The wrist is immobilized for 3-5 days in a volar plaster splint extending from the distal palmar flexion crease to the elbow. Thereafter the splint may be removed periodically (4-5 times daily) to permit active exercise of the wrist. Soaking the hand and forearm for 15-20 minutes in warm water 2-3 times a day tends to relieve pain and stiffness. The splint can usually be discarded within 2 weeks.

B. Marked Displacement: Early reduction and immobilization are indicated. When reduction has been delayed until preliminary healing is advanced, open reduction may be elected or correction of the deformity can be deferred until healing is sound. The malunion can then be corrected by osteotomy and bone grafting.

In an old person with complicating arthritis, when impaction causes stability, the mild deformity may be accepted in favor of early restoration of function.

1. Stable fractures - Colles' fracture is characterized by comminution of the dorsal cortex. Correction of the deformity creates a wedge-shaped area of fragmented cancellous bone. The base of the wedge is directed dorsally, and there is no buttress to prevent recurrence of displacement. In part, stability is attained by bringing the volar cortices of the fragments into anatomic apposition.

Muscular relaxation of the extremity can usually be attained more readily under general anesthesia.

Reduction is by manipulation. After the success of manipulation has been determined by clinical examination, swelling is dissipated by massaging the volar and lateral aspects of the radius until the subcutaneous border can be palpated. Restoration of the normal convex curve of the distal radius implies that transverse displacement and angulation are absent. Proximal displacement has been reduced if the radial styloid process can be palpated 1 cm. distal to the ulnar styloid.

A lightly padded tubular cast extending above the elbow or a "sugar tong" splint (see below) is preferred. The plaster should extend distally only to the palmar flexion crease, with the forearm in midposition and the wrist in slight volar flexion and ulnar deviation. In obese patients, immobilization is more reliable if the elbow is included in the plaster. After the plaster has been applied, it is molded carefully around the wrist until it has set. X-rays are taken while anesthesia is continued. If x-rays show that reduction is not adequate, remanipulation is carried out immediately.

X-ray examination is repeated on the third day and thereafter at weekly intervals during the first 3 weeks. The plaster must remain snug; if loosening occurs after absorption of hemorrhagic exudate, a new cast should be applied.

2. Unstable fractures - If x-rays show extensive comminution with intra-articular extension and involvement of the volar cortex, the fracture is likely to be unstable and skeletal distraction is probably indicated by means of traction on Kirschner wires (see p. 572). Traction is continued while the plaster is applied. To ensure maximal external support, the extremity is immobilized in a tubular plaster cast ex-
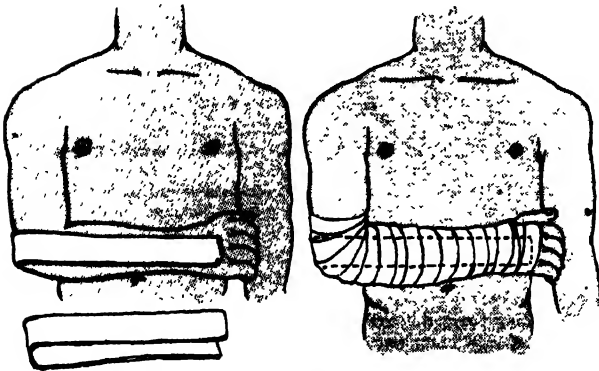
"Sugar Tong" Plaster Splint

Skeletal Distraction for Closed
Manipulation of Fracture
of the Forearm or Wrist
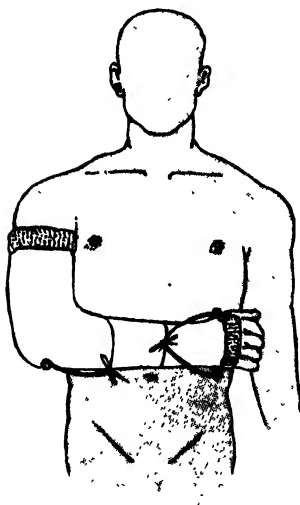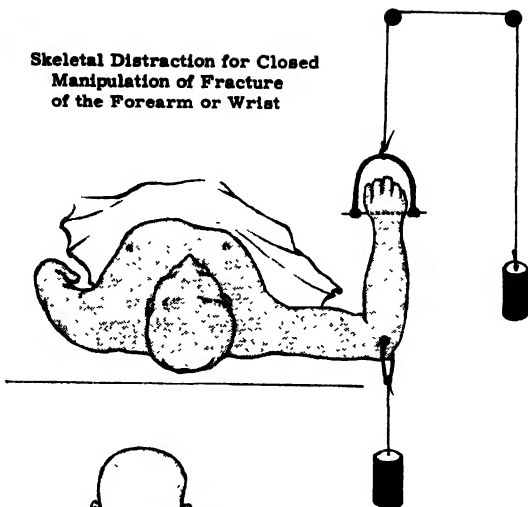
Full Upper Extremity
Plaster With Kirschner
Wires Incorporated

tending from the axilla to the palmar flexion crease, with the
elbow at a right angle, the forearm in midposition, and the
wrist in slight ulnar deviation and volar flexion. The Kirsch-
ner wires are incorporated in the plaster, and the bows are
maintained to hold the wires taut (see above). The wires are
left in place for 6 weeks. The fracture is protected for an
additional 2 weeks following removal of the wires by a
gauntlet or a "sugar tong" splint.

Postreduction Treatment.

Frequent observation and careful management can prevent or minimize some of the disabling sequelae of Colles' fracture. The patient's full cooperation in the exercise program is essential. If comminution is marked, if swelling is severe, or if there is evidence of median nerve deficit, the patient should remain under close observation (preferably in a hospital) for at least 72 hours. The extremity should be elevated to minimize swelling, and the adequacy of circulation determined at frequent intervals. Active exercise of the fingers and shoulder is encouraged. In order that the extremity should be used as much as possible, the plaster should be trimmed in the palm to permit full finger flexion.

As soon as the plaster is removed, the patient is advised to use the extremity for customary daily care but to avoid possible refracture.

Complications and Sequelae.

Joint stiffness is the most disabling sequel of Colles' fracture. Derangement of the distal radioulnar joint may be caused by the original injury or by incomplete reduction; it is characterized by restriction of forearm movements and pain (see p. 575). Late rupture of the extensor pollicis longus tendon is relatively uncommon. Symptoms of median nerve injury usually do not persist more than 6 months, but prolonged symptoms can cause the "carpal tunnel syndrome" (see p. 488).

SMITH'S FRACTURE ("REVERSED COLLES")

The distal fragment of the radius is displaced forward and the apex of angulation is directed dorsally so that the normal volar convexity of the lower radius is increased. The ulnar head may be displaced dorsally. Derangement of the inferior radioulnar joint may be associated

The fracture can be reduced by closed manipulation, and immobilized with the wrist in dorsiflexion and ulnar deviation

FRACTURE OF THE RADIAL STYLOID

Forced radial deviation of the hand at the wrist joint can fracture the radial styloid. A large fragment of the styloid is usually displaced by impingement against the carpal navicular. Avulsion of the tip of the styloid by the radial collateral ligament occurs less frequently, and may be associated with dislocation of the radiocarpal joint. If the fragment is large, it can be displaced farther by the brachioradialis muscle, which inserts into it.

Because the fracture is intra-articular, reduction should be anatomic. If the styloid fragment is not displaced, immobilization in a plaster gauntlet for 3 weeks is sufficient. If the fragment is displaced, manipulative reduction should be tried. The wrist is then immobilized in a snugly molded plaster gauntlet for 6 weeks X-ray examination is repeated every week for at least 3 weeks.

If closed methods fail, open reduction is indicated since persistent displacement is likely to cause post-traumatic degenerative arthritis relatively early.

FRACTURE OF THE DISTAL RADIAL EPIPHYSIS

Fracture of the distal radial epiphysis in children is the counterpart of Colles' fracture in the adult. Wrist sprain is rare in childhood and should be differentiated as early as possible from fracture of the distal epiphysis. Such an injury is usually caused by indirect violence due to a fall on the outstretched hand. The magnitude of displacement of the epiphysial fragment varies.

In some cases separation and displacement of the epiphysis cannot be demonstrated by radiologic examination and may be quite difficult to identify on clinical examination. The patient may complain of pain in the region of the wrist joint, and slight swelling may be present. Pressure with a blunt object, e.g., the eraser of a lead pencil, may demonstrate maximal tenderness at the epiphysial line instead of at the wrist joint. Buckling of the adjacent metaphysial cortex manifests greater displacement.

Displacement is posterior and to the radial side. Marked displacement may be accompanied by crushing of the epiphysial line, tear of the triangular fibrocartilage of the distal radioulnar articulation, displacement of the distal ulnar epiphysis, or avulsion of the ulnar styloid.

Both wrists should be examined by x-ray if injury to the distal radial epiphysis is suspected. Severe injury, crushing the epiphysial cartilage and fracturing the epiphysis, is likely to impede growth and may even lead to early epiphysial fusion; continued growth of the distal ulnar epiphysis produces derangement of the distal radioulnar joint.

If radial deviation of the hand becomes marked, symptomatic Madelung's deformity may result.

Open reduction is rarely necessary. The trauma of the operation superimposed on the injury is likely to cause early arrest of epiphysial growth. Closed reduction by manipulation is usually successful if it can be done within the week following injury. Immobilization is with a plaster gauntlet or "sugar tong" splint. The plaster should be worn for 4-6 weeks. Permanent stiffness due to immobilization of the wrist is not to be feared.

The child should be examined yearly for any untoward sequelae.

FRACTURE-DISLOCATIONS OF THE
RADIOCARPAL JOINT

Dislocation of the radiocarpal joint without fracture is rare. Dislocation without injury to 1 of the carpal bones is usually associated with fracture of the anterior surface of the radius or the ulna. Comminuted fracture of the distal radius may involve either the anterior or posterior cortex and may extend into the wrist joint. Subluxation of the carpus may occur at the same time. The most common fracture-dislocation of the wrist joint involves the posterior or anterior margin of the anterior surface of the radius.

Anterior Fracture-Dislocation of the Radiocarpal Joint (Barton's Fracture).
Anterior fracture-dislocation of the wrist joint is characterized by fracture of the volar margin of the carpal articular surface of

the radius. The fracture cleft extends almost proximally in the coronal plane in an oblique direction, so that the free fragment has a wedge-shaped configuration. The carpus is displaced volar and proximally with the articular fragment. This uncommon injury should be differentiated from Smith's fracture by x-ray examination.

Treatment by closed reduction may be successful, but the instability caused by the configuration of the fracture cleft may necessitate skeletal distraction. Immobilization is with a tubular plaster cast extending from the palmar flexion crease to above the elbow with the wrist in dorsiflexion and the elbow at a right angle. Immediate x-rays are taken in 2 projections. If reduction is not anatomic, skeletal distraction may be necessary. Weekly x-ray examination should oe repeated during the first month. Skeletal distraction should be continued for 6 weeks or until preliminary bone healing has stabilized the fracture.

Posterior Fracture-Dislocation of the Radiocarpal Joint.

Posterior fracture-dislocation of the wrist joint should be differentiated from Colles' fracture by x-ray. In most cases the marginal fragment is smaller than in anterior injury, and often involves the medial aspect where the extensor pollicis longus crosses the distal radius; if reduction is not anatomic, fraying of the tendon at this level may lead to late rupture.

Treatment is by manipulative reduction as for Colles' fracture and immobilization in a snug plaster gauntlet.

DISLOCATION OF THE DISTAL RADIOULNAR JOINT

The triangular fibrocartilage is the most important structure in preventing dislocation of the distal radioulnar joint; complete anterior or posterior dislocation implies a tear of the triangular fibrocartilage and disruption of accessory joint ligaments. The accessory ligaments and the pronator quadratus muscle play a secondary role. Tearing of the triangular fibrocartilage in the absence of major injury to the supporting capsular ligaments causes subluxation or abnormal laxity of the joint. Since the ulnar attachment of the triangular fibrocartilage is at the base of the styloid process, x-rays may demonstrate fracture. Widening of the cleft in comparison with the opposite radioulnar joint suggests diastasis

Complete anterior or posterior dislocation of the distal radioulnar joint is rare. Medial dislocation is associated with fracture of the radius. The direction of dislocation is indicated by the location of the ulnar head in relation to the distal end of the radius.

FRACTURES AND DISLOCATIONS OF THE CARPUS

Injury to the carpal bones occurs predominantly in men during the most active period of life. Because it is difficult to differentiate these injuries by clinical examination, it is imperative to obtain x-ray films of the best possible quality. The oblique film should be taken in midpronation, the anteroposterior film with the wrist in maximal ulnar deviation. Special views, such as midsupination to demonstrate the pisiform, and carpal tunnel views for the hamate, may be necessary.

Fracture of the Carpal Navicular.

The most common injury to the carpus is fracture of the navic- ular. Fracture of the carpal navicular should be suspected in any injury to the wrist in an adult male unless a specific diagnosis of another type of injury is obvious. If tenderness on the radial aspect of the wrist is present and fracture cannot be demonstrated, initial treatment should be the same as if fracture were present (see below) and should be continued for at least 3 weeks. Further x-ray exam- ination after 3 weeks may demonstrate an occult fracture.

Three types of fracture are distinguished:

A. Fracture of the Tubercle: This fracture usually is not widely displaced, and healing is generally prompt if immobilization in a plaster gauntlet is maintained for 2-3 weeks.

B. Fracture Through the Waist: Fracture through the narrowest portion of the bone is the most common type. The blood supply to the proximal fragment is usually not disturbed, and healing will take place if reduction is adequate and treatment is insti- tuted early. If the nutrient artery to the proximal third is in- jured, avascular necrosis of that portion of the bone may occur

X-ray examination in multiple projections is necessary to determine the direction of the fracture cleft and displacement of the proximal fragment. If the proximal fragment is dis- placed, it can be reduced under local anesthesia by forced dorsiflexion and radial deviation of the wrist. Immobilization in a plaster gauntlet with the wrist in slight dorsiflexion is necessary. The plaster should extend distally to the palmar flexion crease in the hand and to the base of the thumbnail. If reduction has been anatomic and the blood supply to the proximal fragment has not been jeopardized, adequate bone healing can be expected within 10 weeks. However, such healing must be demonstrated by the disappearance of the fracture cleft and restoration of the trabecular pattern between the 2 main frag- ments. X-ray examination should be repeated 3 weeks after removal of the cast to verify healing.

C. Fracture Through the Proximal Third: Fracture through the proximal third of the navicular is likely to be associated with injury to the arterial supply of the minor fragment. This can be manifest by avascular necrosis of that fragment. If the lesion is observed soon after injury, reduction and immobili- zation in a plaster gauntlet will promote healing. The plaster gauntlet should be applied snugly, and must be changed if it becomes loose; it is usually necessary to renew the gauntlet every 4 weeks. X-rays should be taken once a month to deter- mine the progress of bone healing; it may be necessary to pro- long immobilization for 4-6 months. The same criteria of radiographic examination as are used for healing of fractures through the waist are used in fractures of the proximal third It is advisable to make an additional x-ray examination 3-4 weeks after removal of the cast.

If evidence of healing is not apparent after immobilization for 6 months or more, further immobilization will probably not be effective. This is especially true if x-rays show that the fracture cleft has widened and if sclerosis is noted adjacent to the cleft. If the interval between the time of injury and the establishment of a

diagnosis is 3 months or more, a trial of immobilization for 2-3 months may be elected   If obliteration of the fracture cleft and evidence of restoration of bone continuity are not visible in x-rays taken after this trial period, some form of operative treatment will be necessary to initiate bone healing   Bone grafting is probably more successful. Prolonged immobilization in a plaster gauntlet is necessary before bony continuity is restored.

If avascular necrosis has occurred in the proximal fragment, bone grafting is less likely to be successful. Although excision of the avascular fragment may relieve painful symptoms for a time, the patient usually notes weakness of grasp and discomfort after prolonged use. Post-traumatic arthritis is apt to develop late

Prolonged failure of bone healing predisposes to post-traumatic arthritis. Bone grafting operations or other procedures directed toward restoration of bone continuity may be successful, but arthritis causes continued disability. Arthrodesis of the wrist gives the best assurance of relief of pain and a functionally competent extremity.

Fracture of the Lunate.

Fracture of the lunate may be manifested by minor avulsion fractures of the posterior or anterior horn. Careful x-ray examination is necessary to establish the diagnosis. Either of these lesions may be treated by the use of a volar splint for 3 weeks.

Fracture of the body may be manifest by a crack or by comminution. A fissure fracture can be treated by immobilization in plaster for 3 weeks

One complication of this fracture is persistent pain in the wrist, slight restriction of motion, and tenderness over the lunate. X-ray examination can demonstrate areas of sclerosis and rarefaction. Collapse can be accompanied by arthritic changes surrounding the lunate. This x-ray appearance is referred to as Kienböck's disease, or avascular necrosis.

Fracture of the Hamate.

Fracture of the hamate may occur through the body, and is shown on x-ray as a fissure or compression. Fracture of the base of the hamulus is less common and more difficult to diagnose; special projections are necessary to demonstrate the cleft. If the hamulus is displaced, closed manipulation will not be effective. Prolonged painful symptoms or evidence of irritation of the ulnar nerve may require excision of the loose fragment.

Fracture of the Triquetrum.

Fracture of the triquetrum is caused commonly by direct violence and is often associated with fracture of other carpal bones Treatment is by immobilization in a plaster gauntlet for 4 weeks.

Dislocation of the Lunate.

Dislocation of the lunate is the second most common injury of the carpus. Dislocation is caused by forced dorsiflexion of the wrist, and the direction of dislocation is almost always anterior. The diagnosis is usually made by x-ray examination. Dislocation may be manifest by dorsal displacement of the capitate while the lunate retains contact with the radius. A further degree of injury

is manifest by complete displacement of the lunate from the radius, so that it comes to lie anterior to the capitate and loses its relationship to the articular surface of the radius. If x-ray examination is adequate, the diagnosis can be established easily.

Reduction may be achieved by closed manipulation. X-rays are then taken to determine the success of treatment. If reduction is adequate, the extremity is immobilized in a plaster gauntlet with the wrist in volar flexion for about 2 weeks. The plaster is then removed and another applied with the wrist in neutral position. If further x-rays show that this manipulative maneuver has not caused reduction, skeletal distraction by means of Kirschner wires is indicated (see p. 572).

Dislocation of the Capitate.

Dislocation of the capitate is most often associated with other lesions of the carpus, and is commonly called transcarpal or midcarpal dislocation. The most frequent accompanying injury is fracture of the carpal navicular, in which case the lunate and the proximal fragment of the navicular retain their relationship to the articular surface of the radius whereas the distal fragment of the navicular, the capitate, and the remainder of the carpus are displaced dorsally. Thus the dislocation is retrolunar.

Subluxation of the navicular is less often associated with dislocation of the lunate. The direction of dislocation is retrolunar. Fracture of the radioulnar styloid processes is likely to be present also.

INJURIES OF THE HIP REGION

BIRTH FRACTURE OF THE UPPER FEMORAL EPIPHYSES

Birth injury to the upper femoral epiphyses is rare, and the diagnosis is difficult because the skeletal structures involved are deeply situated. Swelling of the upper thigh and pseudoparalysis of the extremity following a difficult delivery suggest the injury. X-ray examination may demonstrate outward and proximal displacement of the shaft of the femur. Formation of new bone in the region of the metaphysis may be demonstrated in 7-10 days. If displacement has occurred, treatment for 2-3 weeks by Bryant's traction (see p. 588) is recommended. Otherwise, protection in a pillow splint for the same length of time is adequate.

DISPLACEMENT AND SEPARATION OF THE CAPITAL FEMORAL EPIPHYSIS

Displacement of the capital femoral epiphysis due to trauma in the normal child must be differentiated from slipped epiphysis (epiphysiolysis, adolescent coxa vara) due to endocrine or metabolic dysfunction. However, in children between the ages of 10 and 16 years, if displacement has been caused by severe trauma, the differentiation may not be possible.

Traumatic separation of the capital femoral epiphysis is rare in normal children, but it may occur as a result of a single episode of severe trauma which otherwise might cause fracture of the femoral neck. The direction of displacement is the same as in adolescent coxa vara. If the patient is examined soon after injury, treatment may be by closed manipulation and internal fixation. Anteroposterior and lateral x-rays are then taken with the extremity in extension and internal rotation. If x-rays show that reduction is anatomic, internal fixation should be done immediately. Although the patient may be ambulatory on crutches, weight-bearing is not permitted until the epiphysial line is fused and avascular necrosis is not likely to occur   Full weight-bearing is not allowed for 3-6 months.

FRACTURE OF THE FEMORAL NECK

Fracture of the femoral neck occurs most commonly in patients over the age of 50. If displacement has occurred, the extremity is in external rotation and adduction. Leg shortening is usually obvious. Motion of the hip joint causes pain. If the fracture is impacted in the valgus position, the injured extremity may be slightly longer than the opposite side and active external rotation may not be possible. If the fragments are not displaced and the fracture is stable, pain at the extremes of passive hip motion may be the only significant finding. The fact that the patient can actively move the hip often obscures the diagnosis

Before treatment is instituted, anteroposterior and lateral films of excellent quality must be obtained; if adequate detail is not demonstrated in the initial films, repeat exposures should be made after a trial of traction and gentle internal rotation of the extremity.

Fractures of the femoral neck may be classified as abduction or adduction fractures.

Abduction Fracture of the Femoral Neck.

Abduction describes the relationship between the neck and shaft fragment and the head, which creates a coxa valga deformity. Abduction fractures occur most often in the proximal femoral neck adjacent to the head. Displacement is apt to be minimal, and impaction is often present. The direction of the fracture cleft approaches the transverse plane of the body, and the angle is 30° or less (see p. 580). The anteroposterior x-ray may show a wedge-shaped area of increased density whose base is directed superiorly. A good lateral film will demonstrate both the anterior and posterior cortices of the femoral neck. In this plane the neck and shaft fragment may be angulated slightly, so that only the posterior cortex appears to be impacted and the anterior cortices of the fragments appear to be separated.

Impaction is precarious and undependable as a fixation mechanism; if internal fixation is not used, separation may occur before healing is sound. If firm impaction can be demonstrated in both the anterior and posterior x-rays, some surgeons recommend conservative treatment, i.e., bed rest with the extremity in balanced suspension for 4-8 weeks. The patient is then permitted to be ambulatory with crutches, but full weight-bearing is not permitted

until complete healing can be demonstrated by x-rays (usually 4-12 months after injury). Other surgeons prefer internal fixation because it permits the patient to be out of bed soon after the operation, even though unsupported weight-bearing is not permitted any sooner than after nonoperative treatment.

If examination in the lateral projection does not show firm impaction of both cortices at the fracture site, closed manipulation and internal fixation are indicated; if angulation is only in the transverse plane, it should be corrected if possible so that the cortices will be in contact.

Adduction Fracture of the Femoral Neck.

Adduction fracture is characterized by coxa vara deformity of the upper femur. The fracture may be at any level of the neck, and the direction of the fracture cleft approaches that of the sagittal plane of the body. Displacement is usually present. The relatively vertical configuration of the fracture cleft favors proximal displacement of the distal fragment by the force of any axial thrust transmitted through the extremity. The fracture should be considered unstable if the angle between the fracture cleft and the transverse plane of the body is greater than 30° (Pauwels) (see below). Internal fixation is necessary to prevent displacement after reduction.

Pauwels' Angles
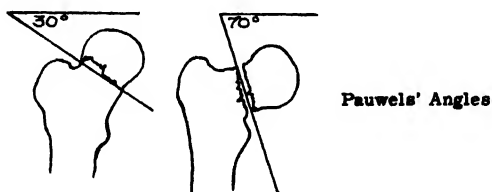
Adduction fracture of the femoral neck is a life-endangering injury; treatment is directed toward the preservation of life and restoration of function of the hip joint. Some surgeons believe that immediate operative treatment is required; others reply that 1-2 days of evaluation in traction is associated with a lowered mortality rate.

Preliminary treatment may be by skin or skeletal traction. X-rays are repeated in 12-24 hours. Comminution which was not evident on the initial examination will often be demonstrated on films taken with the fracture in a more anatomic position.

Open operation is the treatment of choice. If internal fixation is adequate, immobilization in a plaster spica is not necessary. The patient can be free in bed and can be mobilized at an early date, and complications are minimized.

Some surgeons believe that anatomic reduction is desirable; others feel that slight over-reduction is more desirable. The advocates of over-reduction maintain that relative coxa valga increases the stability of the fracture after reduction.

Leadbetter's test for adequacy of reduction consists of placing the heel on the palm of the surgeon's hand with the hip and knee in

extension. If the extremity does not fall into external rotation, it is assumed that torsional displacement has been corrected and the fragments are impacted. Before further operative treatment is undertaken, the test must be validated by anteroposterior and lateral x-rays.

If reduction is adequate, internal fixation is indicated (without exposure of the fracture site) to immobilize the fragments so that the patient can use the extremity during healing.

If adequate reduction cannot be obtained by closed manipulation, open reduction is necessary.

The best functional results are achieved when the fracture heals in anatomic position. Avascular necrosis may be caused by destruction of blood supply to the capital fragment from tearing of the retinacular vessels.

If fixation is precarious, traction in balanced suspension for 1-4 months may be necessary until preliminary healing gives additional stability (see p. 582). Otherwise the patient may be free in bed and can be lifted into a wheel chair once or twice daily soon after operation. Weight-bearing should not be permitted until bone continuity is restored and avascular necrosis is no longer a threat. The agile and cooperative patient may be ambulatory on crutches (but without weight-bearing) within a few days after operative treatment. Crutch walking is hazardous in elderly patients.

The most common sequelae of cervical fracture of the femur are redisplacement after reduction, failure of bone healing, and avascular necrosis of the head fragment. Post-traumatic arthritis appears relatively late

Femoral Neck Fracture in Childhood.

Traumatic cervical fracture of the femur in childhood (rare) must be differentiated from congenital coxa vara. Traumatic fracture is usually caused by severe injury. Anatomic reduction by closed manipulation and immobilization in a plaster spica are necessary to prevent deformity. Internal fixation with wires or screws may be necessary Removal of the fixation apparatus after healing may prevent early fusion of the epiphysis.

Avascular necrosis of the capital epiphysis is a frequent sequel.

TROCHANTERIC FRACTURES

Fracture of the Lesser Trochanter.

Isolated fracture of the lesser trochanter is quite rare but may occur as a result of the avulsion force of the iliopsoas muscle or as a component of intertrochanteric fracture.   •

Fracture of the Greater Trochanter.

Isolated fracture of the greater trochanter may be caused by direct injury, or may occur indirectly as a result of the activity of the gluteus medius and gluteus minimus muscles. It occurs most commonly as a component of intertrochanteric fracture.

If displacement is less than 1 cm. and there is no tendency to further displacement, treatment may be by bed rest with the lower extremity in abduction for 4 weeks. Weight-bearing is permitted as soon as healing is apparent, usually in 6-8 weeks. If displace-
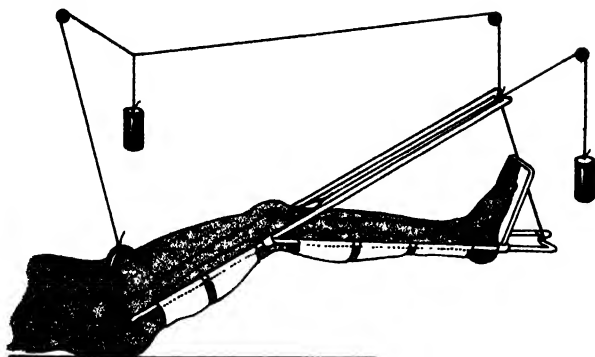
Above: Method of Suspension of Lower Extremity With Skeletal Traction on Tibial Tubercle
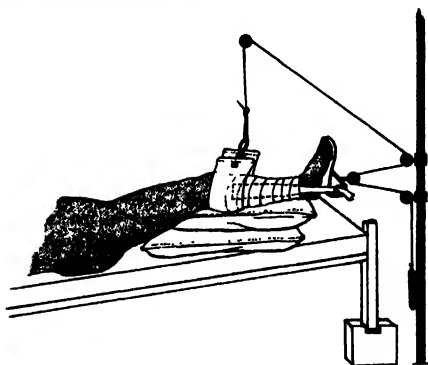
Right: Russell's Traction
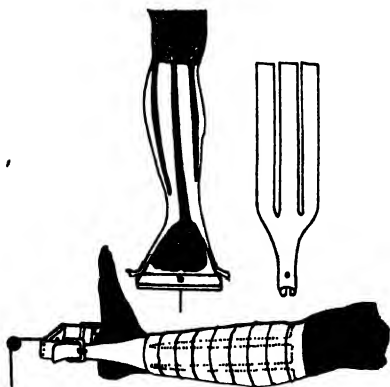
Left: Method of Application of Skin Traction

ment is greater than 1 cm. and increases on adduction of the thigh, extensive tearing of surrounding soft tissues may be assumed and open reduction is indicated, followed by internal fixation with 2-3 loops of stainless steel wire.

Intertrochanteric (Including Pertrochanteric) Fractures.

These fractures occur most commonly among elderly persons. The cleft of an intertrochanteric fracture extends upward and outward from the medial region of the junction of the neck and lesser trochanter toward the summit of the greater trochanter. Petrochanteric fracture includes both trochanters, and is likely to be comminuted.

It is important to determine whether comminution has occurred and the magnitude of displacement. These fractures may vary from fissure fracture without significant separation to severe comminution into 4 major fragments: head-neck, greater trochanter, lesser trochanter, and shaft. Displacement may be so marked that the head-neck fragment forms a right angle with the shaft fragment and the distal fragment is rotated externally through an arc of 90°.

Undisplaced fracture can often be treated by balanced suspension of the lower extremity. If displacement is present, traction is necessary in addition to balanced suspension. If the general condition of the patient is satisfactory, a choice can be made between operative treatment and continued closed treatment. If reduction is satisfactory and immobilization is adequate, bone healing usually occurs without internal fixation.
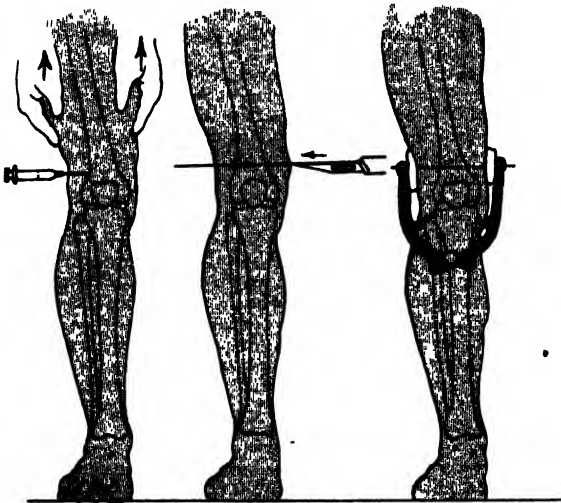
Technic of Insertion of Kirschner Wire in the Supracondylar Region of the Femur

If the fracture cleft is only a fissure and separation is insig-nificant, treatment may be by balanced suspension for 2-3 months. Weight-bearing should not be resumed until the fracture cleft has been obliterated by healing.

If comminution is present and displacement is significant, balanced suspension and skeletal traction by means of a Kirschner wire through the tibial tubercle may be tried.

If closed methods are not successful, open operation is neces-sary. However, it is extremely difficult to reduce comminuted fractures by open methods and the operation should not be under-taken lightly. If adequate reduction can be obtained either by closed or open methods, internal fixation of the fracture is justifiable to prevent further displacement.

Intertrochanteric fracture during childhood can be treated by skeletal traction with a Kirschner wire inserted through the lower femur above the epiphysial line (see p. 583) or by closed reduction and internal fixation. Varus deformity should be avoided if possible. The incidence of avascular necrosis of the capital femoral epiphysis following this fracture is high.

SUBTROCHANTERIC FRACTURE

Subtrochanteric fracture due to severe trauma occurs below the level of the lesser trochanter at the junction of cancellous and cortical bone. It is most common in men during the active years of life. Soft tissue damage is extensive. The direction of the fracture cleft may be transverse or oblique. Comminution occurs occasionally and may extend proximally into the intertrochanteric region or distally into the shaft. Muscle is often interposed be-tween the major fragments.

Closed reduction should be attempted by continuous traction to bring the distal fragment into alignment with the proximal fragment. If comminution is not extensive and the lesser trochanter is not de-tached, the proximal fragment is often drawn into relative flexion, external rotation, and abduction by the activity of the iliopsoas, gluteus medius, and gluteus minimus muscles.

Prolonged skeletal traction by means of a Kirschner wire in-serted through the supracondylar region of the femur (with the hip and knee flexed to a right angle) is necessary (see p. 585). X-ray is repeated every 12-24 hours. If soft tissue interposition is not a factor, reduction can be achieved within 48 hours. Thereafter the extremity is left in this position with an appropriate amount of traction until stabilization occurs, usually in 4-8 weeks. The angle of flexion is then reduced by gradually bringing the hip and knee into extension. After 2-3 months of continuous traction the extremity can be immobilized in a plaster spica. Weight-bearing must not be resumed for 6 months or even longer, until bone healing obliterates the fracture cleft.

Interposition of soft tissue between the major fragments may prevent closed reduction. Open reduction of this fracture is diffi-cult and should be undertaken early; if treatment is delayed until the third week following injury, extensive bleeding at the fracture site is likely to be encountered.

Method of Suspension of
    Lower Extremity With
    Skeletal Traction for
    Subtrochanteric
    Fracture
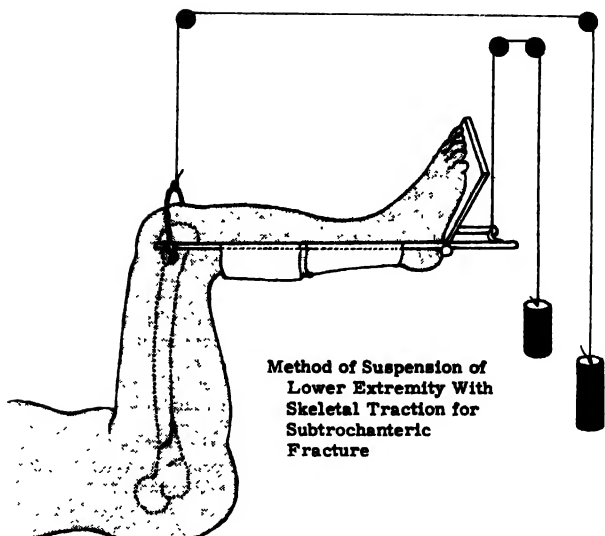
After open reduction has been performed, internal fixation is required to prevent redisplacement. If comminution is present, or if it is important to avoid prolonged immobilization, biplane fixation is recommended.

The activity status after operation depends upon the adequacy of internal fixation. If fixation is precarious, skeletal traction in balanced suspension should be continued until healing is well under way. Otherwise the patient may be ambulatory on crutches (but without weight-bearing) a few days following surgery.

DISLOCATION OF THE HIP JOINT

Dislocation of the hip joint may occur with or without fracture of the pelvis. It occurs most commonly during the most active years of life as a result of severe trauma. The femoral head can be completely dislocated from the acetabulum only if the ligamentum teres is ruptured.

Posterior Hip Dislocation.
The head of the femur is usually dislocated posterior to the acetabulum while the thigh is flexed, e.g., as may occur in a head-on automobile collision when the passenger's knee is driven violently against the dashboard.

The significant clinical findings are shortening, adduction, and internal rotation of the extremity. Anteroposterior, transpelvic, and, if fracture of the acetabulum is demonstrated, oblique projec-

tions are required. Common complications are fracture of the
acetabulum, injury to the sciatic nerve, and fracture of the head
or shaft of the femur. The head of the femur may be displaced
through a rent in the posterior hip joint capsule, or the glenoid lip
may be avulsed from the acetabulum. The short external rotator
muscles of the hip joint are commonly lacerated. Fracture of the
posterior margin of the acetabulum creates an unstable mechanism.

If the acetabulum is not fractured or if the fragment is small,
reduction by closed manipulation either by Bigelow's or Stimson's
method is indicated.

The success of reduction is determined immediately by antero-
posterior and lateral x-rays. Interposition of capsule substance
will be manifest by widening of the joint cavity. If reduction is
adequate the hip will be stable with the extremity in extension and
slight external rotation.

Postreduction treatment may be by immobilization in a plaster
spica or by balanced suspension. Since this is primarily a soft
tissue injury, sound healing should take place in 3 weeks. Active
hip joint movements may be instituted then, but even partial weight-
bearing should be deferred for at least 3 months and full weight-
bearing for 6 months.

If the posterior or superior acetabulum is fractured, disloca-
tion of the hip must be assumed to have occurred even though dis-
placement is not present at the time of examination. Undisplaced
fissure fractures may be treated by bed rest for 3 weeks and
avoidance of weight-bearing for 2 months. Frequent examination
is necessary to make certain that the head of the femur has not be-
come displaced from the acetabulum.

Minor fragments of the posterior margin of the acetabulum
may be disregarded unless they are in the hip joint cavity. Larger
displaced fragments often cannot be reduced adequately by closed
methods. If the fragment is large and the hip is unstable following
closed manipulation, open operation is indicated. If the sciatic
nerve has been injured it should be exposed and treated by appro-
priate neurosurgical technics when the posterior hip joint is ex-
posed. The fragment is then placed in anatomic position and fixed
with 1-2 bone screws.

After the operation the patient is placed in bed with the extrem-
ity in balanced suspension (see p. 582) under 10-15 lb. of skeletal
traction on the tibial tubercle for about 6 weeks or until healing of
the acetabular fracture is sound. Full weight-bearing is not per-
mitted for 6 months or more.

Anterior Hip Dislocation.

, In anterior hip dislocation the head of the femur may lie medi-
ally on the obturator membrane, beneath the obturator externus
muscle (obturator or thyroid dislocation), or, in a somewhat more
superior direction, beneath the iliopsoas muscle and in contact
with the superior ramus of the pubis (pubic dislocation). The thigh
is classically in flexion, abduction, and external rotation, and the
head of the femur is palpable anteriorly and distal to the inguinal
flexion crease. Anteroposterior and lateral films are required.

Closed manipulation under general anesthesia is usually ade-
quate.

Postreduction treatment may be by balanced suspension or by
immobilization in a plaster spica with the hip in extension and the

extremity in neutral rotation. Active hip joint motion is permitted after 3 weeks.

Central Dislocation of the Hip With Fracture of the Pelvis.

Central dislocation of the head of the femur with fracture of the acetabulum may be caused by crushing injury or by an axial force transmitted through the abducted extremity to the acetabulum. Comminution is commonly present. There are usually 2 fragments: superiorly, the ilium; inferiorly and medially, the obturator ring. Fracture occurs near the roof of the acetabulum, and the components of the obturator ring are displaced inward with the head of the femur. Extensive soft tissue injury and massive bleeding into the soft tissues are likely to be present. Intra-abdominal injury must not be overlooked. Stereoanteroposterior and oblique x-rays are required.

In the absence of complicating injury, closed manipulation and skeletal traction under general anesthesia are recommended. Reduction is then verified by x-ray.

Heavy skeletal traction should be continued for 1-2 weeks and then gradually reduced to 15-20 lb. Traction must be continued for about 6 weeks or until stabilization occurs, which may take 8-12 weeks. Suspension is then discontinued and active exercises are given to promote recovery of hip joint motion. Full weight-bearing is not permitted for 6 months.

FRACTURE OF THE SHAFT OF THE FEMUR

FRACTURE OF THE SHAFT OF THE FEMUR IN ADULTS

Fracture of the shaft of the femur usually occurs as a result of severe direct trauma. Most are closed fractures; open fracture is often the result of compounding from within. Extensive soft tissue injury, bleeding, and shock are commonly present.

If the fracture is through the upper third of the shaft, the proximal fragment is apt to be in flexion, external rotation, and abduction, with proximal displacement or overriding of the distal fragment. In midshaft fracture the direction of displacement is not constant, but the distal fragment is almost always displaced proximally if the fracture is unstable; and angulation is commonly present with the apex directed anterolaterally. In complete fracture of the lower third of the shaft the distal fragment is often displaced proximally; the upper end of the distal fragment may be displaced posteriorly to the distal end of the upper fragment.

The most significant features are severe pain in the thigh and deformity of the lower extremity. Surgical shock is likely to be present. Careful x-ray examination in at least 2 planes is necessary to determine the exact site and configuration of the fracture cleft. Splints should be removed either by the surgeon or by a qualified assistant so that manipulation will not cause further injury. The hip and knee should be examined for associated injury.
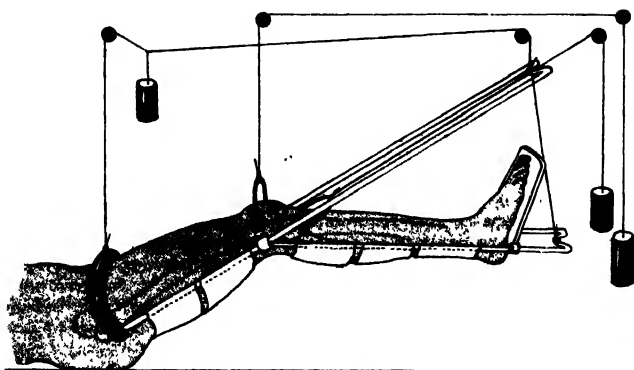
Method of Suspension of Lower Extremity With Biplane Skeletal Traction for Supracondylar Fracture
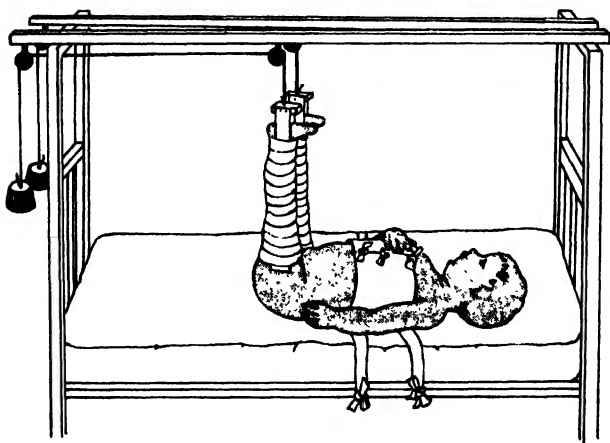
Bryant's Traction

Injuries to the sciatic nerve and to the superficial femoral artery and vein are not common but must be recognized promptly. Surgical shock and secondary anemia are the most important early complications. Later complications are essentially those of prolonged recumbency, e.g., the formation of renal calculi.

Treatment.

Treatment depends upon the age of the patient and the site and configuration of the fracture. Oblique, spiral, and comminuted fractures are unstable, and can rarely be treated successfully by closed manipulation and external plaster fixation. Traction followed by closed manipulation should be tried. Skeletal traction is probably the most effective form of treatment.

After preliminary traction, biplane x-rays are made to determine the progress of correction of overriding. If alignment and apposition of fragments are not satisfactory, closed manipulation, preferably under general anesthesia, should be carried out.

If soft tissue interposition prevents reduction by closed methods, open reduction is required.

  A. Fracture of the Upper Third: The treatment of subtrochanteric fracture is discussed on p. 584. If a comminuted subtrochanteric fracture extends into the upper third of the femoral shaft, it may be necessary to use skeletal traction through the supracondylar region of the femur and suspend the extremity with hip and knee at a right angle (see p. 585). Otherwise, skeletal traction can be through the lower femur on the tibial tubercle with the extremity at a less acute angle in balanced suspension. Russell's traction can be used if the patient is small and muscular development is not great   External rotation and abduction of the extremity are usually required to bring the lower fragment into alignment with the proximal fragment
  B. Fracture of the Middle Third: The deformity caused by fracture at this level is not constant. Angulation is commonly present with the apex directed anterolaterally. Treatment may be by skeletal traction through the tibial tubercle or the lower end of the femur (see p. 582). Traction in the transverse plane by a swathe around the thigh may be necessary to prevent recurrence of angulation.
  C. Fracture of the Lower Third: The proximal end of the distal fragment is likely to be displaced posterior to the distal end of the proximal fragment. The same displacement is likely to be encountered in supracondylar fracture. Russell's traction should not be used since it may injure the femoral or popliteal vessels. Simultaneous skeletal traction through the distal femur at right angles to the tibial tubercle can correct displacement of the distal fragment. The same mechanism of traction is used for supracondylar fracture (see p. 588).

FRACTURE OF THE SHAFT OF THE FEMUR
IN INFANTS AND CHILDREN

Femoral fracture at birth occurs most often in the middle third. Comminution is usually not present.

Skin traction (see p. 588) and plaster immobilization are adequate, although skeletal traction may be necessary in older children.

Open reduction is rarely necessary.

Fracture of the proximal or middle third of the femur in a child under 5 years of age can be treated with Bryant's traction. Adhesive strips are applied to both extremities from the upper thirds of the thighs to the supramalleolar regions, and held firm by circular bandages. Both hips are flexed to a right angle, and the knees are maintained in full extension. Sufficient traction force is applied to raise the buttocks free of the bed. Circulatory adequacy must be observed carefully, and another method substituted if swelling of the foot, cyanosis or pallor of the foot, or obliteration of pedal pulsations cannot be managed by adjustment of dressings.

As a rule, sufficient callus is present at the fracture site after 3-4 weeks so that traction can be discontinued. If callus formation is adequate, infants who have not yet begun to walk need no further protection; walking infants may require a single plaster hip spica for an additional 4-6 weeks.

Preliminary treatment of unstable fracture of the femur in children over 5 years of age can be by Russell's traction. If the child is uncooperative, or if adequate correction (see p. 582) cannot be obtained, it may also be necessary to place the sound extremity in traction. Traction should be continued until the fracture is stabilized; if traction is discontinued before the reparative callus is sufficiently mature, the deformity (especially angulation) may recur even though the extremity is protected by a plaster spica. Correction of angulation and torsional displacement around the long axis of the femur are mandatory. Slight shortening (1-2 cm.) can be compensated by growth. Close apposition of fragments is not necessary, since healing will take place in spite of minimal soft tissue interposition.

It is usually necessary to continue traction for 6-8 weeks or until sufficient callus has formed to prevent recurrence of the deformity. Immobilization in a plaster spica should be maintained for another 2 months. Weight-bearing must not be resumed until x-rays show that healing is sound.

INJURIES OF THE KNEE REGION

FRACTURES OF THE DISTAL FEMUR

Supracondylar Fracture of the Femur.

This comparatively uncommon fracture (at the junction of cortical and cancellous bone) may be transverse, oblique, or comminuted. The distal end of the proximal fragment is apt to perforate the overlying vastus intermedius or vastus medialis muscle, and may penetrate the suprapatellar pouch of the knee joint to cause hemarthrosis. The proximal end of the distal fragment is usually displaced posteriorly and slightly laterally.

Since the distal fragment may impinge upon the popliteal vessels, circulatory adequacy distal to the fracture site should be verified as soon as possible. Absence of pedal pulsations is an indication for immediate reduction. If pulsation does not return promptly after reduction, immediate exploration and appropriate treatment of the vascular lesion is indicated.

A less frequent complication is injury to the peroneal or tibial nerve.

If the fracture is transverse or nearly so, closed manipulation under general anesthesia will occasionally be successful. Stable fractures can be immobilized in a single plaster spica with the hip and knee in about 30° of flexion. Frequent x-ray examination is necessary to make certain that redisplacement has not occurred.

Stable or unstable uncomplicated supracondylar fracture is best treated with biplane skeletal traction if soft tissue interposition does not interfere with reduction (see p. 588). If adequate reduction cannot be obtained, it may be necessary to manipulate the fragments under general anesthesia, using skeletal traction to control the distal fragment.

Traction must be continued for about 6 weeks or until stabilization occurs. The wires can then be removed and the extremity immobilized in a single plaster spica for an additional 2-3 months.

An alternative method of treatment (applicable only to stable fractures) is to incorporate the wires into a plaster spica after reduction.

Supracondylar fracture is likely to be followed by restriction of knee motion.

Intercondylar Fracture of the Femur.

This uncommon comminuted fracture, which occurs only in older patients, is classically described as "T" or "Y" according to the x-ray configuration of the fragments. Closed reduction is difficult when the proximal shaft fragment is interposed between the 2 main distal fragments. Maximal recovery of function of the knee joint requires anatomic reduction of the articular components. If alignment is satisfactory and displacement minimal, immobilization for about 4 months in a plaster spica will be sufficient. If displacement is marked, skeletal traction through the tibial tubercle (with the knee in flexion) is required. Manual molding of the distal fragments may be necessary. Open reduction and bolt fixation of the distal fragments may be indicated to restore articular congruity. Further treatment is as described for supracondylar fracture.

Condylar Fracture of the Femur.

Isolated fracture of the lateral or medial condyle of the femur is a rare consequence of severe trauma. Occasionally only the posterior portion of the condyle is separated. The cruciate ligaments or the collateral ligament of the opposite side of the knee are often injured.

The objective of treatment is restoration of anatomic intraarticular relationships. If displacement is minimal, the knee can be manipulated into varus or valgus (opposite the position of deformity). If anatomic reduction cannot be obtained by closed manipulation, open reduction and fixation of the minor fragment with 2-3 bone screws is recommended. The ligaments must be explored, and repaired if found to be injured.

Separation of the Distal Femoral Epiphysis.

Traumatic separation of the distal femoral epiphysis in children is the counterpart of supracondylar fracture in the adult. The direction of displacement of the epiphysial fragment is most com-

monly anterior. Torsional displacement around the long axis of
the femur may be associated.

Reduction of anterior displacement can be achieved by closed
manipulation. After reduction is complete, the knee is flexed to a
right angle and a tubular plaster cast is applied from the inguinal
region to the toes. If the thigh is obese, the plaster should be ex-
tended proximally to include the pelvis in a single hip spica.

Peripheral circulation must be observed carefully. After 4
weeks the plaster is changed and flexion of the knee reduced to 135°
At the end of the second month the patient may be permitted to be
free in bed until he regains complete knee extension.

FRACTURES OF THE PROXIMAL TIBIA

Fracture of the Lateral Tibial Condyle.

Fracture of the lateral condyle of the tibia is commonly caused
by a blow on the lateral aspect of the knee with the foot in fixed
position, producing an abduction strain. Hemarthrosis is always
present, as the fracture cleft involves the knee joint. Soft tissue
injuries are likely to be present also. The tibial collateral and
anterior cruciate ligaments may be torn. A displaced free frag-
ment may tear the overlying lateral meniscus. If displacement is
marked, fracture of the proximal fibula may be present also.

The objective of treatment is to restore the articular surface
and normal anatomic relationships, so that torn ligaments can heal
without elongation. In cases of minimal displacement where liga-
ments have not been extensively damaged, treatment may be by
immobilization for 6 weeks in a tubular plaster cast extending from
the toes to the inguinal region with the knee in slight flexion. Re-
duction of marked displacement can be achieved by closed manipu-
lation unless comminution is severe. After x-ray verification of
reduction, the extremity can be immobilized in a tubular plaster
cast extending from the inguinal region to the toes, preferably with
the knee in full extension.

Many fractures of the lateral condyle of the tibia, especially
comminuted fractures, cannot be reduced adequately by closed
methods. Open reduction and stabilization with a bolt or multiple
bone screws may be necessary.

Fracture of the Medial Tibial Condyle.

Fracture of the medial condyle of the tibia is caused by the
adduction strain produced by a blow against the medial aspect of
the knee with the foot in fixed position. The medial meniscus and
the fibular collateral ligament may be torn. Severe comminution
is not usually present, and there is only 1 major free fragment.

Treatment is by closed reduction to restore the articular sur-
face of the tibia so that ligamentous healing can occur without
elongation. After reduction the extremity is immobilized for 10-12
weeks in a tubular plaster cast extending from the inguinal region
to the toes with the knee in full extension. Weight-bearing is not
permitted for 4 months.

Fracture of Both Tibial Condyles.

Axial force, such as may result from falling on the foot or
sudden deceleration with the knee in full extension (during an auto-

mobile accident), can cause simultaneous fracture of both condyles of the tibia. Comminution is apt to be severe. Swelling of the knee due to hemarthrosis is marked. Deformity is either genu varum or genu valgum. X-ray examination should include oblique projections.

Severe comminution makes anatomic reduction difficult to achieve by any means and difficult to maintain following closed manipulation alone. Sustained skeletal traction is usually necessary. When stability has been achieved, the extremity can be immobilized for another 4-6 weeks in a tubular plaster cast extending from the toes to the inguinal region with the knee in full extension. Unassisted weight-bearing is not permitted before the end of the fourth month.

If closed methods are not effective, open reduction must be attempted.

Instability and restriction of motion of the knee are common sequelae. If reduction is not adequate, post-traumatic arthritis will appear early.

Fracture of the Tibial Tuberosity.

Violent contraction of the quadriceps muscle may cause avulsion of the tibial tuberosity. Avulsion of the anterior portion of the upper tibial epiphysis, uncommon in childhood, must be differentiated from Osgood-Schlatter disease (osteochondrosis of the tibial tuberosity).

When avulsion of the tuberosity is complete, active extension of the knee is not possible.

If displacement is minimal, treatment is by immobilization in a tubular plaster cast extending from the inguinal to the supramalleolar region with the knee in full extension. Immobilization is maintained for 8 weeks or until stabilization occurs.

A loose fragment which has been displaced more than 0.5 cm. can be treated either by closed reduction and percutaneous fixation, with plaster immobilization, or by open reduction.

Fracture of the Tibial Spine.

This injury usually occurs in association with comminuted fracture of the condyles. The anterior tibial spine may be avulsed with the anterior cruciate ligament. Hemarthrosis is always present. Instability of the knee can be demonstrated by forward displacement of the upper end of the tibia when the knee is flexed to a right angle (anterior "drawer" sign).

Undisplaced fracture may be treated by immobilization of the extremity for 6 weeks in a tubular plaster cast extending from the inguinal region to the toes with the knee in slight flexion. In displaced fracture it is necessary to reposition the fragment in anatomic position by arthrotomy of the knee. The fragment is secured by a wire loop passed through a drill hole and attached to the anterior cortex of the tibia. The knee is then immobilized for about 8 weeks as above.

If the lesion is not recognized until 4-8 weeks after the injury, restriction of knee joint extension may be the most significant finding. In adults, excision of the fragment and reinsertion of the ligament may be necessary to permit recovery of function.

Separation of the Proximal Tibial Epiphysis.

Complete displacement of the proximal tibial epiphysis is rare; partial separation due to forceful hyperextension of the knee is more common. The distal metaphysial fragment is displaced posteriorly.

If no circulatory or neurologic deficit is present, immediate treatment by closed manipulation is indicated. Anteroposterior films are then exposed by holding the plate against the anterior surface of the leg with the beam directed through the lower thigh. A lateral film is also prepared. If x-rays show that reduction is adequate, a heavy anterior plaster splint is applied from the inguinal region to the bases of the toes and maintained in position with circular bandages around the foot, ankle, and upper thigh. The bandages are wrapped in figure-of-eight fashion around the thigh and leg (as is done for supracondylar fracture of the humerus). Peripheral circulation must be observed frequently for at least 72 hours. After 3 weeks the knee may be brought to a right angle and a tubular plaster cast applied from the inguinal region to the toes for another 3-4 weeks. Even guarded weight-bearing must be avoided until the end of the second month.

Injury to the proximal tibial epiphysial plate as a result of a blow on the lateral aspect of the knee causes compression but only minor displacement. This lesion, analogous to fracture of the lateral condyle in the adult, is apt to retard or arrest the growth of the lateral aspect of the tibia and thus cause tibia valga, or knock-knee. Surgical arrest of the growth of the medial tibial epiphysis may be necessary to prevent this deformity.

FRACTURE OF THE PROXIMAL FIBULA

Isolated fracture of the proximal fibula is uncommon, and is usually associated with fracture of the femur or of the tibia or fracture-dislocation of the ankle joint. The apex of the fibular head may be avulsed by the activity of the biceps femoris muscle or detached with the fibular collateral ligament by an adduction strain.

The fracture usually requires no treatment, but avulsion of the apex of the head may necessitate operative repair of the ligament or tendon.

Fracture in this region may be associated with paralysis of the common peroneal nerve.

DISLOCATION OF THE PROXIMAL TIBIOFIBULAR JOINT

This extremely rare lesion is caused by the activity of the biceps femoris muscle. Displacement is posterior, and can be reduced by digital pressure over the head of the fibula in the opposite direction.

FRACTURE OF THE PATELLA

Transverse Fracture of the Patella.

Transverse fracture of the patella is the result of indirect violence, usually with the knee in semiflexion. Fracture may be due

to sudden voluntary contraction of the quadriceps muscles or sudden forced flexion of the leg when these muscles are contracted. The level of fracture is most often in the middle. The extent of tearing of the patellar retinacula depends upon the degree of force of the initiating injury. The activity of the quadriceps muscles causes displacement of the proximal fragment; the magnitude of displacement is dependent upon the extent of the tear of the quadriceps expansion.

Swelling of the anterior knee region is caused by hemarthrosis and hemorrhage into the soft tissues overlying the joint. If displacement is present, the defect in the patella can be palpated and active extension of the knee is lost.

Open reduction is indicated if the fragments are separated more than 2-3 mm. The fragments must be accurately repositioned to prevent early post-traumatic arthritis of the patellofemoral joint. If the minor fragment is small (no more than 1 cm. in height), it may be excised and the rectus or patellar tendon (depending upon which pole of the patella is involved) sutured directly to the major fragment. If the fragments are approximately the same size, repair by wire cerclage is preferred.

Removal of 50% of the patella causes incongruity of joint surfaces, and post-traumatic arthritis may occur early.

Comminuted Fracture of the Patella.

Comminuted fracture of the patella is caused only by direct violence. Little or no separation of the fragments occurs because the quadriceps expansion is not extensively torn. Severe injury may cause extensive comminution of the articular cartilages of both the patella and the opposing femur. If comminution is not severe and displacement is insignificant, plaster immobilization for 8 weeks in a cylinder extending from the groin to the supramalleolar region is sufficient.

Severe comminution requires excision of the patella and repair of the defect by imbrication of the quadriceps expansion.

TEAR OF THE QUADRICEPS TENDON

Tear of the quadriceps tendon occurs most often in patients over 40 years of age. Preexisting attritional disease of the tendon is apt to be present, and the causative injury may be minor. The tear commonly results from sudden deceleration, such as stumbling, or slipping on a wet surface. A small flake of bone may be avulsed from the superior pole of the patella, or the tear may occur entirely through tendinous tissue.

Pain may be noted in the anterior knee region. Swelling is due to hemarthrosis. The patient is unable to extend his knee completely. X-rays may show avulsion of a bit of bone from the superior patella.

Operative repair is required. If treatment is delayed until partial healing has occurred, the suture line can be reinforced by transplantation of the iliotibial band from the upper extremity of the tibia.

TEAR OF THE PATELLAR LIGAMENT

The same mechanism which causes tears of the quadriceps tendon may also cause tear of the patellar ligament, transverse fracture of the patella, or avulsion of the tibial tubercle. The characteristic clinical finding is proximal displacement of the patella. A bit of bone may be avulsed from the lower pole of the patella.

Operative treatment is necessary. The ligament is resutured to the patella and any tear in the quadriceps expansion is repaired. The extremity should be immobilized for 8 weeks in a tubular plaster case extending from the inguinal to the supramalleolar region. Guarded exercises may then be started.

DISLOCATION OF THE PATELLA

Traumatic dislocation of the patellofemoral joint is most often associated with dislocation of the knee joint When this injury occurs alone it is usually due to direct violence and the direction of dislocation of the patella may be lateral. Spontaneous reduction is apt to occur if the knee joint is extended, if so, the clinical findings may consist merely of hemarthrosis and localized tenderness over the medial patellar retinaculum. Gross instability of the patella, which can be demonstrated by physical examination, indicates that injury to the soft tissues of the medial aspect of the knee has been extensive. Operative repair is necessary.

DISLOCATION OF THE KNEE JOINT

Traumatic dislocation of the knee joint is uncommon in adults and extremely rare in children. It is caused by severe trauma. Displacement may be transverse or torsional Complete dislocation can occur only after extensive tearing of the supporting ligaments, and is apt to cause injury to the popliteal vessels or the tibial and peroneal nerves.

Signs of neurovascular injury below the site of dislocation are an absolute indication for prompt reduction under general anesthesia, since failure of circulation will undoubtedly result in gangrene of the leg and foot. Axial traction is applied to the leg and a shearing force is exerted over the fragments in the appropriate direction. If pedal pulses do not return promptly, the popliteal fossa should be explored at once.

, Anatomic reduction of uncomplicated dislocation should be attempted. If impinging soft tissues cannot be removed by closed manipulation, arthrotomy is indicated. After reduction the extremity is immobilized in a tubular plaster cast extending from the inguinal region to the toes with the knee in slight flexion. (In the obese patient a single hip spica should be applied.) A window should be cut in the plaster over the dorsum of the foot to allow frequent inspection of dorsalis pedis pulsation. After 8 weeks' immobilization the knee can be protected by a long leg brace. Intensive quadriceps exercises are necessary to minimize functional loss.

INTERNAL DERANGEMENTS OF THE KNEE JOINT

Internal derangements of the knee joint mechanism may be caused by trauma or attritional disease. Although ligamentous and cartilaginous injuries are discussed separately, they may occur as combined lesions.

Injury to the Menisci.

Injury to the medial meniscus is the most frequent internal derangement of the knee joint. Any portion of the meniscus may be torn. A marginal tear permits displacement of the medial fragment into the intercondylar region ("bucket-handle tear"). A fragment of cartilage displaced between the articular surfaces of the femur and tibia prevents either complete extension or complete flexion.

The significant clinical findings are swelling (due to hemarthrosis) and varying degrees of restriction of flexion or extension. Motion may cause pain over the anteromedial or posteromedial joint line. Tenderness can often be elicited at the point of pain. Forcible external rotation of the foot with the knee flexed to a right angle may cause pain over the medial joint line. If symptoms have persisted for 2-3 weeks, weakness and atrophy of the quadriceps femoris may be present.

Injury to the lateral meniscus less often causes mechanical blockage of joint motion. Pain and tenderness may be present over the lateral joint line. Pain can be elicited by forcible rotation of the leg with the knee flexed to a right angle.

Initial treatment may be conservative. Swelling and pain can be relieved by aspiration. If pain is severe, the extremity should be immobilized in a posterior plaster splint with the knee in slight flexion. Younger patients usually prefer to be ambulatory on crutches, but immediate weight-bearing must not be permitted. As long as acute symptoms persist, quadriceps exercises should be performed frequently throughout the day with the knee in maximum extension (as a "straight leg lift"). Unrestricted activity must not be resumed until complete motion is recovered and healing is complete.

Exploratory arthrotomy is advisable for recurrent "locking," recurrent effusion, or disabling pain. Quadriceps exercises are instituted immediately after the operation and gradually increased in frequency. As soon as the patient is able to perform these exercises comfortably, graded resistance maneuvers should be started. Exercises must be continued until all motion has been recovered and the volume and competency of the quadriceps are equal on both sides.

Injury to the Collateral Ligaments.

The collateral ligaments prevent excursion of the joint beyond normal limits. When the knee is in full extension, the collateral ligaments are taut; in flexion, only the anterior fibers of the tibial collateral ligament are taut.

A. Tibial Collateral Ligament: Forced abduction of the leg at the knee causes injury varying from tear of a few fibers to complete rupture of the ligament. A bit of bone may be avulsed from its femoral or tibial attachment.

A history of a twisting injury at the knee with valgus strain can usually be obtained. Pain is present over the medial aspect of the knee joint. In severe injury the intra-articular fluid may be increased. Tenderness can be elicited at the site of the lesion. X-ray examination of an isolated lesion is not helpful unless it is made under stress. Under local or general anesthesia the extremities are bound together in full extension at the knee joint, and an anteroposterior film is made with the legs in forcible abduction. Widening of the medial joint cleft suggests complete rupture.

Treatment of incomplete tear consists of protection from further injury while healing progresses. Hemarthrosis should be relieved by aspiration. The knee may be immobilized in a posterior plaster splint or a tubular cast extending from the inguinal to the supramalleolar region.

Complete rupture should be repaired immediately so that healing will take place without ligamentous elongation and subsequent instability of the knee joint.

B. Fibular Collateral Ligament: Tear of the fibular collateral ligament is often associated with injury to surrounding structures, e.g., the popliteus muscle tendon and the iliotibial band. Avulsion of the apex of the fibular head may occur, and the peroneal nerve may be injured.

Pain and tenderness are present over the lateral aspect of the knee joint, and hemarthrosis may be present. X-rays may show a bit of bone avulsed from the fibular head. If severe injury is suspected, x-ray examination under stress, using local or general anesthesia, is required. A firm, padded nonopaque object about 8-12 inches in diameter is placed between the knees and the legs are forcibly adducted while an anteroposterior exposure is made. Widening of the lateral joint cleft indicates severe injury.

The treatment of partial tear is similar to that described for partial tear of the medial collateral ligament. If complete tear is suspected, and especially if the peroneal nerve has been injured, exploration is indicated. The extremity is protected for 8 weeks in a plaster cylinder extending from the inguinal region to above the ankle.

Injury to the Cruciate Ligaments.

The function of the anterior and posterior cruciate ligaments is to restrict anterior and posterior gliding of the tibia when the knee is flexed. If the tibia is rotated internally on the femur, the ligaments twist around themselves and become taut; if the tibia is rotated externally, they become lax.

A. Anterior Cruciate Ligament: Injury to the anterior cruciate ligament is usually associated with injury to the medial meniscus or the tibial collateral ligament. The cruciate ligament may be avulsed with the anterior tibial spine, or may rupture within the substance of its fibers.

The characteristic clinical sign of tear of either cruciate ligament is a positive "drawer" sign: the knee is flexed at a right angle and pulled forward; if excessive anterior excursion of the proximal tibia can be noted, the anterior ligament is torn.

Complete recent rupture of the anterior cruciate ligament can occasionally be repaired with stout absorbable sutures as described for fixation of the anterior tibial spine. Old tears require reconstructive procedures.

B. Posterior Cruciate Ligament: Tear of the posterior cruciate ligament can be diagnosed by the "drawer" sign: the knee is flexed at a right angle and the upper tibia is pushed backward; if excessive posterior excursion of the proximal tibia can be noted, the ligament is torn.

Primary repair is of dubious value. Treatment is directed at the associated injuries and maintenance of the competency of the quadriceps musculature.

FRACTURES OF THE SHAFTS OF THE TIBIA AND FIBULA

Fracture of the shaft of the tibia or fibula may occur at any age. In general, open, transverse, comminuted, and segmental fractures are caused by direct violence; spiral and oblique fractures are caused by indirect violence. Fracture of the middle third of the shaft (especially if comminuted) is apt to be complicated by delay of bone healing.

If fracture is complete and displacement is present, clinical diagnosis is not difficult. However, critical local examination is of utmost importance in planning treatment. The nature of the skin wounds which communicate with the fracture site indicate the type of compounding injury. A small laceration without contused edges suggests that the point of a bone fragment has caused compounding from within. A large wound with contused edges, especially over the subcutaneous surface of the tibia, suggests compounding from without. The presence of abrasions more than 6 hours old, blebs, pyoderma, and preexisting ulcers precludes immediate open treatment of closed fracture. Extensive swelling due to hemorrhagic exudate in closed fascial compartments may prevent immediate reduction. Edematous and hemorrhagic infiltration may prevent adequate closure of a surgical incision. Neurovascular integrity below the level of the fracture must be verified before definitive treatment is instituted.

Anteroposterior and lateral projections of the entire leg, including both the knee and ankle joints, are always necessary, and oblique projections are often desirable. The surgeon must know the exact site and configuration of the fracture, the severity of comminution, and the direction of displacement of fragments.

FRACTURE OF THE SHAFT OF THE FIBULA

Isolated fracture of the shaft of the fibula is usually caused by direct trauma. If no other lesion is present, immobilization for 4 weeks in a plaster boot (equipped with a walking surface) extending from the knee to the toes is sufficient. Complete healing is the rule.

FRACTURE OF THE SHAFT OF THE TIBIA

Fracture of the shaft of the tibia is stable unless the fibula is fractured also. Marked displacement does not usually occur, and overriding is not significant unless there is associated dislocation of either tibiofibular joint.

If the fragments are not displaced, or if more than 75% of their transverse diameter remains in contact, treatment may be by immobilization in a tubular plaster cast extending from the inguinal region to the toes with the knee in about 30° of flexion and the foot in neutral position. The plaster should be changed 2-4 weeks after the injury to correct the loosening which will occur as a result of absorption of hemorrhagic exudate and atrophy of the thigh and calf muscles. Immobilization should be continued for at least 10 weeks, or until healing is demonstrated by x-ray.

If the fragments are displaced, manipulation under anesthesia may be necessary. Fractures with a transverse cleft tend to be unstable. Oblique and spiral fractures tend to become displaced unless the fragments are locked.

A tubular plaster cast is applied as for undisplaced fracture. If x-rays do not show satisfactory apposition of fragments, alternative methods of treatment should be used (see below).

FRACTURE OF THE SHAFTS OF BOTH
BONES IN ADULTS

Fracture of the shafts of the tibia and fibula are unstable lesions which tend to become displaced following reduction. Treatment is directed toward reduction and stabilization of the tibial fracture until healing takes place. For adequate reduction the fragments must be apposed almost completely, and angulation and torsional displacement of the tibial fracture must be corrected.

If reduction by closed manipulation is anatomic and angular and torsional displacement are corrected, transverse fractures tend to be stable. Repeated x-rays are necessary to determine whether displacement has recurred. The plaster must remain snug at all times. Recurrent angular displacement can be corrected by dividing the plaster circumferentially and inserting wedges in the appropriate direction. If apposition is disturbed, another type of treatment must be substituted.

If oblique and spiral fractures are unstable following manipulation and immobilization, internal fixation or skeletal traction is usually required (see p. 601). This can be accomplished by closed reduction and skeletal distraction. While traction is continued, a tubular plaster cast with pins or wires incorporated is applied.

An alternative method is continuous skeletal traction. Traction must be continued for about 6 weeks until preliminary healing causes stabilization. The extremity is then immobilized in plaster for at least 12 weeks until bone continuity has been restored.

If adequate apposition and correction of the deformity cannot be achieved by closed methods, open reduction and internal fixation are required.

The blood supply to intermediate fragments is likely to be disturbed in comminuted and segmental fractures. These unstable fractures can usually be treated successfully only by closed reduc-
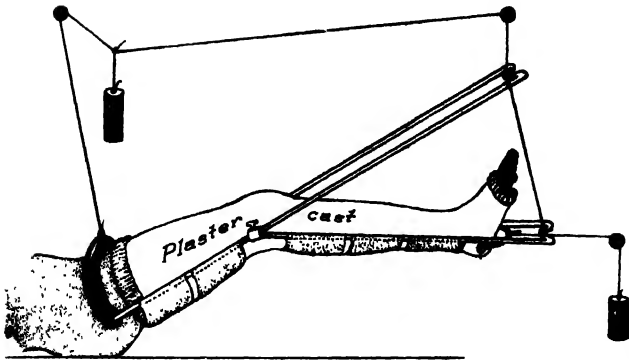
Calcaneal Skeletal Traction and Full Lower Extremity Plaster for
Unstable Fracture of Tibia and Fibula

Left: Weight
Bearing
Plaster Boot
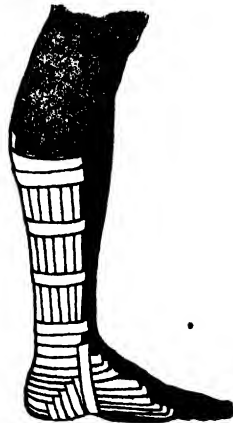
Right: Gibney
Ankle
Strapping

tion and external immobilization. If the patient wishes to remain
ambulatory, percutaneous fixation of the major fragments with
Steinmann pins or Kirschner wires will maintain reduction and
alignment. However, continuous skeletal traction in plaster with
a Kirschner wire inserted through the calcaneus is usually preferred
until stabilization occurs (see above).

FRACTURE OF THE SHAFTS OF THE TIBIA
AND/OR FIBULA IN CHILDREN

Open reduction and internal fixation of closed fractures of the tibia or fibula in children are rarely necessary. If a tibial fracture is stable or if the fibular shaft is intact, closed reduction is indicated after angular and torsional displacement have been corrected. If proper alignment is secured, 1 cm. of overriding is acceptable.

Comminuted or oblique tibial fractures, or fractures of both bones, require continuous skeletal traction by means of a Kirschner wire inserted through the calcaneus, followed by immobilization in a tubular plaster cast until stabilization occurs (usually in 3-4 weeks).

INJURIES OF THE ANKLE REGION

ANKLE SPRAIN

Sprain of the ankle joint during childhood is rare. In the adult, ankle sprain is most often caused by forced inversion of the foot, as may occur in stumbling on uneven ground. Pain is usually maximal over the anterolateral aspect of the joint; greatest tenderness is apt to be found in the region of the anterior talofibular and talocalcaneal ligaments. Eversion sprain is less common; maximal tenderness and swelling are usually found over the deltoid ligament.

Sprain is differentiated from major partial or complete ligamentous tears by anteroposterior, lateral, and 30° internal oblique x-ray projections; if the joint cleft between either malleolus and the talus is greater than 4 mm., major ligamentous tear is probable. Occult lesions can be demonstrated by x-ray examination under inversion or eversion stress after infiltration of the area of maximal swelling and tenderness with 5 ml. of 2% procaine.

If swelling is marked, elevation of the extremity and avoidance of weight-bearing for a few days is advisable. The ankle can be supported with a Gibney strapping (see p. 601). Adhesive support for another 2 weeks will relieve pain and swelling. Further treatment may be by warm foot baths and elastic bandages. Continue treatment until muscle strength and full joint motion are recovered.

FRACTURES AND DISLOCATIONS OF THE ANKLE JOINT

Fractures and dislocations of the ankle joint may be caused by direct injury, in which case they are apt to be comminuted and open; or by indirect violence, which often causes typical lesions (see below).

Pain and swelling are the prominent clinical findings. Deformity may or may not be present. X-rays of excellent technical quality must be prepared in a sufficient variety of projections to demonstrate the extent and configuration of all major fragments. Special oblique projections may be required.

Fracture of the Medial Malleolus.

Fracture of the medial malleolus may occur as an isolated lesion of any part of the malleolus (including the tip), or may be associated with (1) fracture of the lateral malleolus with medial or lateral dislocation of the talus, and (2) dislocation of the inferior tibiofibular joint with or without fracture of the fibula. Isolated fracture does not usually cause instability of the ankle joint.

Undisplaced isolated fracture of the medial malleolus should be treated by immobilization in a plaster boot extending from the knee to the toes with the ankle flexed to a right angle and the foot slightly inverted to relax the tension on the deltoid ligament (see p. 601). Immobilization must be continued for 6-8 weeks or until bone healing is sound.

Displaced isolated fracture of the medial malleolus may be treated by closed manipulation under general or local anesthesia. The essential maneuver consists of anatomic realignment by digital pressure over the distal fragment, followed by immobilization in a plaster boot (as for undisplaced fracture) until bone healing is sound (see p. 601). If anatomic reduction cannot be obtained by closed methods, open reduction and internal fixation with 1-2 bone screws are required.

Fracture of the Lateral Malleolus.

Fracture of the lateral malleolus may occur as an isolated lesion, or may be associated with fracture of the medial malleolus, tear of the deltoid or posterior lateral malleolar ligament, or avulsion of the posterior tibial tubercle. If the medial aspect of the ankle is injured, lateral dislocation of the talus is apt to be present. The tip of the lateral malleolus may be avulsed by the calcaneofibular and anterior talofibular ligaments. Transverse or oblique fracture may occur. Oblique fractures commonly extend downward and anteriorly from the posterior and superior aspects.

If swelling and pain are not marked, isolated undisplaced fracture of the lateral malleolus may be treated by Gibney ankle strapping (see p. 601). Otherwise, a plaster boot should be applied for 6 weeks and an elastic bandage worn thereafter until full joint motion is recovered and the calf muscles are functioning normally.

Isolated displaced fracture of the lateral malleolus should be treated by closed manipulation. The foot should be immobilized in slight inversion, which tautens the ligaments over the lateral aspect of the ankle joint and tends to prevent displacement.

If anatomic reduction cannot be achieved by closed methods, open reduction is required.

Combined Fracture of the Medial and Lateral Malleoli.

Bimalleolar fracture may be accompanied by medial or lateral dislocation of the talus. In unstable fractures, displacement is apt to recur after closed reduction.

Bimalleolar fracture may be treated by closed manipulation. A tubular plaster cast is then applied from the inguinal region to the toes with the knee in about 45° of flexion and the foot in neutral position. Immediate open reduction must be resorted to if x-rays show that perfect anatomic reduction has not been achieved by closed manipulation.

Fractures of the Distal Tibia.

Fracture of the distal tibia is usually associated with other lesions.

A. **Fracture of the Posterior Margin:** Fracture of the posterior articular margin may involve part or all of the entire posterior half and is apt to be accompanied by fracture of either malleolus and posterior dislocation of the talus. It must be differentiated from fracture of the posterior tibial tubercle, which is usually caused by avulsion with the attached posterior lateral malleolar ligament.

Anatomic reduction by closed manipulation is required if the fracture involves more than 25% of the articular surface. The extremity is immobilized in a plaster cast extending from the inguinal region to the toes with the knee in about 40° of flexion, the ankle at a right angle, and the foot in neutral position.

Frequent x-ray examination is necessary to make certain that redisplacement does not occur. The plaster should be changed as soon as loosening becomes apparent. Immobilization must be maintained for at least 8 weeks. Weight-bearing must not be resumed until bone healing is sound, usually in about 12 weeks.

B. **Fracture of the Anterior Margin:** Fracture of the anterior articular margin of the tibia (rare) is likely to be caused by forced dorsiflexion of the foot. If displacement is marked and the talus is dislocated, tear of the collateral ligaments or fractures of the malleoli are likely to be present.

Reduction is by closed manipulation. If comminution is present, the extremity should be immobilized for about 12 weeks. Healing is apt to be slow.

C. **Comminuted Fractures:** Extensive comminution of the distal tibia ("compression type" fracture) presents a difficult problem of management. Anatomic restoration by open reduction usually is not possible, and the congruity of articular surfaces cannot be restored completely by closed manipulation. The best form of treatment is skeletal traction (see p. 601). A tubular plaster cast is applied from the inguinal region to the toes with the knee in 10-15° of flexion and the foot in neutral position.

Healing is likely to be slow. Traction must be continued for 8-12 weeks or until stabilization occurs. Disabling post-traumatic arthritis is likely to occur early. If the articular surfaces of the ankle joint have not been properly realigned, early arthrodesis is indicated to shorten the period of disability.

Dislocation of the Ankle Joint.

A. **Complete Dislocation:** The talus cannot be completely dislocated from the ankle joint unless all ligaments are torn. This lesion is rare.

B. **Incomplete Dislocation:** Major ligamentous injuries in the region of the ankle joint are usually associated with fracture.

1. **Tear of the deltoid ligament** - Complete tear of the talotibial portion of the deltoid ligament can permit interposition of the posterior tibial tendon between the medial malleolus and the talus. Associated injury is usually present, especially

fracture of the lateral malleolus with lateral dislocation of the talus.

Pain, tenderness, swelling, and ecchymosis in the region of the medial malleolus without fracture suggest partial or complete tear of the deltoid ligament. If fracture of the lateral malleolus or dislocation of the distal tibiofibular joint is present, the cleft between the malleolus and talus is likely to be widened. If widening is not marked, x-ray examination under stress is necessary.

Interposition of the deltoid ligament between the talus and the medial malleolus often cannot be corrected by closed manipulation. If widening persists after closed manipulation, surgical exploration is indicated so that the ligament can be removed and repaired by suture.

Associated fracture of the fibula can be treated by fixation with a coaxial intramedullary standard bone screw or a bone nail to assure maintenance of anatomic reduction.

2. Tear of the talofibular ligament - Isolated tear of the anterior talofibular ligament is caused by forced inversion of the foot. X-ray examination under stress may be necessary, using local or general anesthesia. Both feet are forcibly inverted and an anteroposterior film exposed. If tear is incomplete, the talus will be seen to be laterally displaced from the articular surface of the fibula.

Rupture of the anterior talofibular ligament may be associated with tear of the calcaneofibular ligament. Tear of both ligaments may be associated with fracture of the medial malleolus and medial dislocation of the talus.

Instability of the ankle joint, characterized by a history of recurrent sprains, may result from unrecognized tears of the anterior talofibular ligament.

Recent isolated tear of the anterior talofibular ligament or combined tear of the calcaneofibular ligament should be treated by immobilization for 4 weeks in a plaster boot. Associated fracture of the medial malleolus creates an unstable mechanism. Unless anatomic reduction can be achieved and maintained by closed methods, open reduction of the malleolar fragment is indicated, followed by internal fixation of the fracture and repair of the ligamentous injury.

Dislocation of the Distal Tibiofibular Joint.

Both the anterior and posterior lateral malleolar ligaments must be torn before dislocation of the distal tibiofibular joint can occur. Lateral dislocation of the talus is also an essential feature, and this cannot occur unless the medial malleolus is fractured or the deltoid ligament is torn. The distal fibula is commonly fractured, but it may remain intact, and dislocation may be caused by a tear of the interosseous ligament.

Anatomic reduction by closed manipulation is difficult to achieve, but should be tried. Under general anesthesia, the foot is forced medially by a shearing maneuver and a snug plaster cast is applied from the inguinal region to the toes. If immediate and repeated x-ray examinations do not demonstrate that anatomic reduction has been achieved and maintained, open reduction and internal fixation should be performed as soon as possible.

Separation of the Distal Tibial and Fibular Epiphyses.
    The most common injury of the ankle region of children is traumatic separation of the distal tibial and fibular epiphyses. Sprain is rare in children. Separation of the distal fibular epiphysis may occur as an isolated injury, or may be associated with separation of the tibial epiphysis.
    If displacement has occurred, treatment is by closed manipulation and plaster immobilization. Open reduction is seldom justifiable. If injury has been severe, disturbance of growth is likely to follow.

INJURIES OF THE FOOT

FRACTURE AND DISLOCATION OF THE TALUS

Dislocation of the Subtalar and Talonavicular Joints.
    Dislocation of the subtalar and talonavicular joints without fracture occasionally occurs. The talocrural joint is not injured. Displacement of the foot can be either in varus or valgus. Reduction by closed manipulation is usually not difficult. Incarceration of the posterior tibial tendon in the talonavicular joint may prevent reduction by closed manipulation. After reduction the extremity should be immobilized in a plaster boot for 4 weeks.

Fracture of the Talus.
    Major fracture of the talus commonly occurs either through the body or through the neck. Indirect injury is usually the cause of closed fracture, and comminution is not usually present. The major proximal or distal fragment may be dislocated.
    Closed uncomminuted fracture of the body of the talus with minimal displacement of fragments is not likely to cause disability if immobilization is continued until bone continuity is restored. If displacement occurs, the proximal fragment is apt to be dislocated from the subtalar and ankle joints. Reduction by closed manipulation can be achieved by traction and forced plantar flexion of the foot. The foot should be immobilized for 8-16 weeks in moderate equinus in a plaster boot until bone continuity has been restored. If reduction is not anatomic, fibrous healing of the fracture may cause post-traumatic arthritis, which necessitates arthrodesis of the ankle and subtalar joints.
    Even though adequate reduction has been obtained by closed manipulation, extensive displacement of the proximal body fragment is likely to be followed by avascular necrosis.
    Fracture of the neck of the talus may be complicated by dislocation of either the body or the neck fragment. Forced dorsiflexion of the foot can cause this injury. If dislocation of the body fragment is complete, reduction by closed manipulation may not be possible. If reduction by closed manipulation is not successful, open reduction should be done as soon as possible to prevent avascular necrosis.
    Fracture of the neck of the talus with dislocation of the proximal body fragment is likely to be complicated by avascular necro-

sis. Avascular necrosis is also apt to complicate closed fracture of the talus through the neck with dislocation of the distal fragment from the subtalar and talonavicular joints. If prompt reduction by closed manipulation is not possible, immediate open reduction is advisable since delay may cause necrosis of overlying skin.

FRACTURE OF THE CALCANEUS

Fracture of the calcaneus is commonly caused by direct trauma. Since this fracture is likely to occur as a result of a fall from a height, fracture of the spine may also be present. Comminution and impaction are characteristic. Minor or fissure fractures are easy to miss on clinical and x-ray examination.

Many types of fracture must be differentiated.

Fracture of the Tuberosity of the Calcaneus.

Isolated fracture of the tuberosity is not common. It may occur in a vertical or horizontal direction. Vertical fracture is not likely to cause sequelae if reduction is anatomic and weight-bearing is deferred until bone healing is sound. Horizontal fracture ("beak" fracture) may be limited essentially to the apophysis or may extend toward the subtalar joint into the substance of the tuberosity. The minor fragment may be displaced by the Achilles tendon.

Reduction can be achieved by skeletal traction and closed manipulation. If adequate reduction cannot be achieved in this way, open reduction is advised.

Fracture of the Sustentaculum.

Isolated fracture of the sustentaculum tali is a rare lesion which may be caused by forced eversion of the foot. It is usually associated with comminuted fracture.

Articular Fracture of the Calcaneus.

Most fractures of the calcaneus involve the body and extend into the subtalar joint. Fissure fractures cause minor disability.

Comminuted fractures are of 2 main types:

  A. Minimal Comminution: In fractures with minimal comminution the fracture cleft extends from the medial aspect of the tuberosity distally and somewhat laterally through the posterior articular facet. The sustentaculum tali and the medial portion of the posterior articular facet are not displaced in relation to the talus. The tuberosity and part of the articular portion of the body of the remaining articular facet are impacted and displaced proximally. If reduction is not anatomic, derangement of the subtalar joint occurs and post-traumatic arthritis can be expected.

  B. Extensive Comminution: Fracture with extensive comminution of the subtalar joint may involve the calcaneocuboid joint as well as the tuberosity. This is a serious injury which may cause major disability in spite of the best treatment.

Anatomic realignment of fragments is not possible. Common sequelae are pain in the subtalar region and weakness of the calf muscles.

Some surgeons advise conservative treatment without reduction, with avoidance of weight-bearing until bone healing has taken place. If painful symptoms occur late, arthrodesis of the subtalar joint is indicated. Other surgeons, notably Hermann and Böhler, advocate closed reduction.

FRACTURE OF THE TARSAL NAVICULAR

Transverse fracture of the body of the tarsal navicular with displacement of the dorsal fragment is a most serious injury. Anatomic reduction cannot be achieved either by closed or open methods. Post-traumatic arthritic pain is likely to occur, and can be mitigated only by arthrodesis.

FRACTURES OF THE METATARSALS

Avulsion of the Base of the Fifth Metatarsal.

The base of the fifth metatarsal may be avulsed by the activity of the peroneus brevis muscle. If the minor fragment is not displaced, adhesive strapping is sufficient. Displaced fracture can be treated by immobilization in a plaster walking boot (see p. 601).

Fractures of the Shafts of the Metatarsals.

Undisplaced fractures of the shafts of the metatarsals cause no permanent disability. Treatment may be by wearing a stiff-soled shoe (with partial weight-bearing) or, if pain is marked, a plaster walking boot (see p. 601).

If reduction is not reasonably accurate, fractures of the metatarsal necks can cause disability. Unstable fracture is treated by sustained skeletal traction using a Kirschner wire inserted through the distal phalanx and attached to a banjo splint by an elastic band to maintain reduction until healing occurs. Operative reduction is occasionally necessary.

Fractures of the Toes.

Fractures of the toes are most commonly caused by direct violence. Comminuted fractures of the distal phalanges require only immobilization. Unstable fractures of the proximal phalanges may require sustained skeletal traction until healing is under way.

Fracture of the Sesamoid Bone of the Great Toe.

Fracture of the sesamoid bones of the great toe is rare, but may occur as a result of a crushing injury. It must be differentiated from partite developmental lesions. Undisplaced fracture requires no treatment other than a foot support or a metatarsal bar. Displaced fracture may require immobilization in a walking plaster boot with the great toe strapped in flexion.

Appendix:
Drug Dosages

Calculation of Dosages for Infants and Children.

The adult dosages given in most references usually apply to persons of "average" size and weight ages 20-60. After age 60, and in debilitated patients, the dosages of certain drugs should be reduced. This applies particularly to skin irritants, narcotics, and depressant drugs. Pediatric dosages also must be individualized.

Calculation of dosages for infants and children:

Clark's Rule: $\dfrac{\text{Weight of child (lb.)}}{150} \times \text{Adult dose} = \text{Child's dose}$

Young's Rule: $\dfrac{\text{Age of child}}{\text{Age} + 12} \times \text{Adult dose} = \text{Child's dose}$

DRUGS FOR GENERAL USE

Nonnarcotic Analgesics.
   A. Acetylsalicylic Acid, U.S.P. (Aspirin, A.S.A.®): 0.3-0.6 Gm. (5-10 gr.) every 3-4 hours p.r.n., best taken with $1/2$-1 glass of water or with food. Occasionally causes gastric upset. Large doses cause tinnitus.
   B. Sodium Salicylate, U.S.P.: 0.3-0.6 Gm. (5-10 gr.) every 3-4 hours p.r.n. Occasionally causes gastric upset. Observe caution in giving sodium to cardiac patients.
   C. Acetylsalicylic Acid Compound (APC, Aspirin Compound, Empirin®): Contains acetylsalicylic acid, 225 mg. ($3^{1}/2$ gr.), acetophenetidin, 150 mg. ($2^{1}/2$ gr.), and caffeine, 30 mg. ($1/2$ gr.).
   D. Acetophenetidin, U.S.P. (Phenacetin): 0.3 Gm. (5 gr.) every 3-4 hours p.r.n. For patients unable to take acetylsalicylic acid. Prolonged use occasionally causes methemoglobinemia.
   E. Dextropropoxyphene and Acetylsalicylic Acid Compound (Darvon® Compound): Acetylsalicylic Acid compound with dextropropoxyphene HCl, N.N.D. (Darvon®), 30 mg. ($1/2$ gr.). 1-2 capsules every 6 hours p.r.n.

Narcotic Analgesics.
   A. Morphine Sulfate, U.S.P.: 8-15 mg. ($1/8$-$1/4$ gr.) orally or subcut. every 3 hours p.r.n. May be given I.V. in 5 ml. of saline for rapid effect. A very useful drug; the advantages of the synthetic "substitutes" over morphine have probably been exaggerated. Contraindicated in increased intracranial pressure, respiratory insufficiency, bronchial asthma, Addison's disease, and hepatic disease.

B. **Meperidine Hydrochloride, U.S.P.** (Demerol®, Dolantin®):
50-100 mg. ($3/4$-$1 1/2$ gr.) orally or I.M. (DO NOT GIVE
SUBCUT.) every 3 hours p.r.n. May be given I.V. slowly in
urgent situations. 0.1 Gm. ($1 1/2$ gr.) is equivalent in analgesic
effect to 8 mg. ($1/8$ gr.) morphine, but meperidine causes less
respiratory and cardiac depression. Does not control severe
pain, especially neurogenic pain, as well as morphine.

C. **Anileridine Hydrochloride, N.N.D.** (Leritine®): 25-50 mg.
($3/8$-$3/4$ gr.) orally, subcut., or I.M. every 4-6 hours p.r.n.
Anileridine is 2.5 times as potent as meperidine.

D. **Methadone Hydrochloride, U.S.P.** (Amidone®, Dolophine®):
2.5-10 mg. ($1/24$-$1/6$ gr.) orally, subcut., or I.M. every 3
hours p.r.n. Not well tolerated orally and somewhat irritating
when given parenterally. Sedative effect is less than that of
morphine.

E. **Dihydromorphinone Hydrochloride, U.S.P.** (Dilaudid®): 2.5
mg. ($1/24$ gr.) orally or 1-4 mg. ($1/60$-$1/16$ gr.) subcut. every
3-4 hours p.r.n. Dihydromorphinone is 5-10 times as potent
(and toxic) as morphine.

F. **Codeine Phosphate, U.S.P.:** 8-60 mg. ($1/8$-1 gr.) orally or
subcut. every 3-4 hours p.r.n. Most often used in combination
with aspirin or aspirin compound. If 60 mg. (1 gr.) is not
effective, a more potent analgesic such as morphine is indi-
cated. Skin reactions have been reported. Prolonged use
causes constipation.

G. **Dihydrohydroxycodeinone Compound** (Percodan®): Each tablet
contains salts of dihydrohydroxycodeinone and homatropine,
and aspirin compound. Give 1 tablet every 6 hours p.r.n.
Percodan Demi® contains half the amounts of dihydrohydroxy-
codeinone and homatropine.

Narcotic Antagonists.

A. **Nalorphine Hydrochloride, U.S.P.** (Nalline®): 5-10 mg. ($1/12$-
$1/6$ gr.) subcut., I.M., or I.V., repeated if necessary every
10-15 minutes to a total of 40 mg. ($2/3$ gr.). Counteracts
respiratory depression due to poisoning with morphine and re-
lated drugs within 10 minutes. May produce withdrawal symp-
toms in addicts.

B. **Levallorphan Tartrate** (Lorfan®): 0.5-1 mg. ($1/120$-$1/60$ gr.)
I.V. If necessary, 1-2 additional 0.5 mg. doses may be given.
Counteracts respiratory depression due to poisoning with
morphine and related drugs within 1 minute. Effect lasts 2-5
hours. Does not impair analgesic effect of narcotic drugs.

Antiallergic Agents.

A. **Antihistamines:**
1. **Diphenhydramine Hydrochloride, U.S.P.** (Benadryl®) - 50
mg. ($3/4$ gr.) orally 3-4 times daily, or 5-20 mg. ($1/12$-$1/3$
gr.) I.V. May be given with drug to which allergic reaction
is anticipated. May cause drowsiness.
2. **Tripellenamine Hydrochloride, U.S.P.** (Pyribenzamine®) -
50 mg. ($3/4$ gr.) orally 3-4 times daily.

B. **Adrenergic Drugs:**
1. **Epinephrine Injection, U.S.P.** (1:1000 aqueous solution), or

Sterile Suspension, U.S.P. (1:500 in oil); Adrenaline Injection, B.P. - For immediate response, give 0.2-0.5 ml. of 1:1000 subcut. or I.M. In emergencies give 0.1-0.5 ml. 1:1000 aqueous solution I.V. For prolonged action, give 0.2-1 ml. of 1:500 in oil I.M. every 10-14 hours. Used in treatment of acute allergic reactions or when antihistamines fail to relieve symptoms.

2. Ephedrine Hydrochloride, N.F., or Ephedrine Sulfate, U.S.P. - 25-50 mg. (3/8-3/4 gr.) orally q.i.d.

C. Corticotropin (ACTH) and Steroids: For use in acute or chronic allergic reactions which fail to respond to more conservative measures.

1. Corticotropin, U.S.P. (ACTH) - 5-40 U.S.P. units in any I.V. fluid over 8-12 hours, or 40-200 U.S.P. units I.M. in saline solution every 6 hours. If foreign protein reactions occur or if resistance develops after prolonged use, steroids may be substituted.

2. Prednisone and prednisolone - These delta derivatives of cortisone and hydrocortisone, respectively, have a greater degree of anti-inflammatory and antiallergic activity than their parent compounds. They have no significant sodium-retaining effect but potentially have the same hazardous effects as the cortisones. Prolonged administration of high doses may cause profound metabolic abnormalities. Sudden withdrawal may cause adrenal insufficiency.

   a. Oral preparations - Prednisone, N.N.D. (Deltasone®, Meticorten®), or Prednisolone, N.N.D. (Delta-Cortef®, Hydeltra®), 5-50 mg. (avg. 10-20 mg.) daily orally in divided doses every 4-8 hours.

   b. Parenteral preparations (for I.M. or I.V. use) -
      (1) Prednisolone hemisuccinate (Meticortelone®), 50-100 mg. daily I.M. or I.V.
      (2) Prednisolone-21-phosphate (Hydeltrasol®), 40-100 mg. daily I.M. or I.V.
      (3) Methylprednisolone sodium succinate (Solu-Medrol®), 40-120 mg. daily I.M. or I.V.

3. Hydrocortisone Sodium Succinate, N.N.D. (Solu-Cortef®) - 100-250 mg. I.V. in not less than 30 seconds for immediate effect. May be repeated p.r.n. at increasing intervals (1, 3, 6, 10 hours, etc.). The same precautions must be observed as with prednisone and prednisolone. May also be used in the treatment of adrenal insufficiency.

Sedatives and Hypnotics.

A. Phenobarbital, U.S.P.: 15-30 mg. (1/4-1/2 gr.) orally 2-4 times daily, or 100-200 mg. (1 1/2-3 gr.) h.s. Use cautiously in renal insufficiency. Phenobarbital Sodium, U.S.P., 65-100 mg. (1-1 1/2 gr.) may be given subcut. or I.M.

B. Pentobarbital Sodium, U.S.P. (Nembutal®): 30 mg. (1/2 gr.) orally 3-4 times daily, or 100-200 mg. (1 1/2-3 gr.) h.s. Use cautiously in hepatic insufficiency. May be given I.V. or I.M., 100-250 mg. (1 1/2-4 gr.)

C. Amobarbital Sodium, U.S.P. (Amytal Sodium®): 15-30 mg. (1/4-1/2 gr.) orally 3-4 times daily, or 200-300 mg. (3-5 gr.)

h. s. Use cautiously in hepatic insufficiency. May be given
I. M. or I.V. (as anticonvulsant), 65-600 mg. (1-7$^{1}/2$ gr.) Stat.
D. **Secobarbital Sodium, U.S.P. (Seconal®):** 15-30 mg. ($^{1}/4$-$^{1}/2$
gr.) orally every 4 hours, or 100-200 mg. (1$^{1}/2$-3 gr.) h. s.
Contraindicated in hepatic insufficiency.
E. **Chloral Hydrate, U.S.P.:** 0.25-1 Gm. (3$^{3}/4$-15 gr.) orally
(2.5-10 ml. of 10% solution or 1-2 Noctec® capsules). Usually
diluted with milk or fruit juice. May dilute with 30-60 ml. of
olive oil and give rectally.
F. **Paraldehyde, U.S.P.:** 4-16 ml. orally in fruit juice or milk.
May also give rectally, 16-32 ml. in 30-60 ml. of oil; or I.M.,
5-10 ml. deeply in the buttocks. The stock solution is sterile
and can be used for parenteral as well as oral administration.
I.V. use may cause respiratory arrest or pulmonary edema
and is not recommended.

CARDIORESPIRATORY DRUGS

Coronary Dilator Drugs.
A. **Glyceryl Trinitrate, U.S.P. (Nitroglycerin):** Place one 0.3,
0.4, or 0.6 mg. ($^{1}/200$, $^{1}/150$, or $^{1}/100$ gr.) tablet under the
tongue and allow to dissolve. Begin with 0.3 mg. ($^{1}/200$ gr.)
and increase p.r.n. effectiveness. May repeat or give larger
dose if no response appears in 2-5 minutes. Undesirable side
effects include flushing, pounding pulse, dizziness, and
headache.
B. **Amyl Nitrate, U.S.P.:** 1 pearl, crushed and inhaled, acts in
about 10 seconds. Side effects as for glyceryl trinitrate, above.
C. **Pentaerythritol Tetranitrate, N.N.D. (Peritrate®, etc.):** 10-
20 mg. ($^{1}/6$-$^{1}/3$ gr.) t.i.d., a.c., or 80 mg. (1$^{1}/4$ gr.) as
sustained-action tablet once daily. A long-acting nitrate for
prevention of angina but not for treatment of the acute attack.

Anticoagulants.
A. **Heparin Sodium, U.S.P.:** The optimal dose of heparin is that
which prolongs the Lee-White clotting time to 20-30 minutes.
The clotting time should be determined before each dose or,
with continuous administration, every 4-6 hours. Antidotes
are rarely necessary because the duration of action of heparin
is short. Anticoagulant action may be counteracted immediate-
ly with hexadimethrine (Polybrene®) or protamine sulfate, I.V.,
mg. for mg. of heparin.
1. **I.V. administration** - 25-75 mg. ($^{3}/8$-1$^{1}/4$ gr.) every 4 hours
or as continuous drip of 200 mg. (3 gr.)/L. of I.V. fluid at
a rate of 1 ml./minute. For extracorporeal by-pass pro-
cedures, give 1.5-2 mg. ($^{1}/40$-$^{1}/30$ gr.)/Kg. body weight.
2. **Subcut. administration** (concomitantly with I.V. dosage) -
Use 200 mg. (3 gr.)/ml. concentration. Give 200 mg. (3
gr.)/100 lb. body weight, 250 mg. (4 gr.)/150 lb., 300 mg.
(5 gr.)/200 lb. through a No. 25 needle 1 inch below the iliac
crest in the subcutaneous fat every 12-16 hours.
B. **Prothrombin Depressants:** The dosage schedules given below
are based on the assumption that the prothrombin time is normal.

The objective of therapy is to maintain a prothrombin time of 10-30%. As a rule the prothrombin time is determined before the next dose is administered and the quantity adjusted accordingly. Spontaneous hemorrhages may occur with prothrombin times below 10-20% and should be treated by (1) fresh whole blood transfusion; (2) Phytonadione, U.S.P. (vitamin K_1, Mephyton®), 50-150 mg. ($3/4$-$2^{1}/2$ gr.)(usually 50 mg.) slowly I.V.; or (3) Menadione Sodium Bisulfite, U.S.P. (Hykinone®), 50-100 mg. ($3/4$-$1^{1}/2$ gr.) I.V.

1. Bishydroxycoumarin, U.S.P. (Dicumarol®) - Optimal effect may not be achieved for 72 hours. The initial dose is 300 mg. (5 gr.) orally. In 24 hours, determine prothrombin time and give a second dose; in 72 hours, determine prothrombin time and give a third dose. 200 mg. (3 gr.) is usually required on the second day, and 100 mg. ($1^{1}/2$ gr.) on the third day. Maintain by the fourth day on 25-150 mg. ($3/8$-$2^{1}/2$ gr.) daily or less.

2. Warfarin Sodium, N.N.D. (Coumadin®) - A satisfactory level is usually achieved in 21-24 hours. The initial dose is 1 mg. ($1/60$ gr.)/Kg. body weight (not to exceed 75 mg.) orally or I.V. Maintain on 5-20 mg. ($1/12$-$1/3$ gr.) (usually 10 mg.) daily in 1 dose.

3. Phenindione, N.N.D. (Hedulin®, Danilone®) - Dosage is similar to that of bishydroxycoumarin except that the drug is given in divided doses b.i.d.

Diuretics.

CAUTION: Patients receiving large doses of diuretics, especially over prolonged periods, should be observed for potassium depletion and given supplementary potassium in the diet or as potassium chloride p.r.n.

A. Mercurial Diuretics: These are the most effective and most dependable diuretics available. Response to oral administration is uncertain. Dosage is individualized by response. If time permits, begin with one-half the usual dosage and gauge dosage to maintain a diuresis of 1.5-2 lb. daily. In emergencies, the entire dose may be given Stat. In occasional resistant patients the usual dose may have to be repeated on the first day to initiate diuresis. Premedication with an acidifying diuretic such as ammonium chloride increases the diuretic effect of the mercurials. Allergic reactions and stomatitis may occur.

1. Meralluride Injection, U.S.P. (Mercuhydrin Sodium®) - 1-2 ml. I.V., I.M., or subcut. daily or p.r.n. Meralluride tablets for oral administration and meralluride suppositories are available but are rarely used.

2. Mercaptomerin Sodium, Sterile, U.S.P. (Thiomerin Sodium®) - 1-2 ml. of 50% solution I.V., I.M., or subcut.

3. Mersalyl and Theophylline Injection, U.S.P. (Salyrgan-Theophylline®) - Supplied as ampuls containing 10% mersalyl and 5% theophylline. Give 1-2 ml. I.V., I.M., or subcut. Tablets containing 80 mg. ($1^{1}/4$ gr.) mersalyl and 40 mg. ($2/3$ gr.) theophylline are also available. Give 1 tablet daily.

B. **Acidifying Diuretics:** Ammonium Chloride, U.S.P., 1-2 Gm. (15-30 gr.) q.i.d. for 3-4 days and discontinue for 3-4 days. Used also as premedication to potentiate the action of the mercurial diuretics.

C. **Carbonic Anhydrase Inhibitors:** Acetazolamide, U.S.P. (Diamox®), 0.25-0.5 Gm. (4-7$\frac{1}{2}$ gr.) 2-3 days a week in 1 or 2 doses daily. Effectiveness declines with prolonged use.

D. **Thiazide Derivatives:** Observe carefully for potassium deficiency.
   1. Chlorothiazide, U.S.P. (Diuril®) - 0.25-0.5 Gm. (4-7$\frac{1}{2}$ gr.) 1-4 times daily orally.
   2. Hydrochlorothiazide, U.S.P. (Esidrix®, Hydro-Diuril®) - 25-50 mg. ($\frac{3}{8}$-$\frac{3}{4}$ gr.) daily or b.i.d.

Expectorants.
A. **Glyceryl Guaiacolate** (Robitussin®): 100-200 mg. (1$\frac{1}{2}$-3 gr.) orally every 2-3 hours. Also available with codeine, 10 mg. ($\frac{1}{6}$ gr.) per 5 ml. as Robitussin® A-C.

B. **Potassium Iodide Solution**, N.F.: 0.5 ml. q.i.d. (p.c. and h.s.), gradually increasing to 1-2 ml. q.i.d. Gastric intolerance or symptoms of iodism (coryza, acneform eruptions, etc.) occasionally occur.

C. **Hydriodic Acid Syrup**, N.F.: 0.5 ml. q.i.d. (p.c. and h.s.).

D. CO_2 at intervals by face mask.

Cough Depressant Drugs.
A. **Codeine Phosphate**, U.S.P.: 8-15 mg. ($\frac{1}{8}$-$\frac{1}{4}$ gr.) orally every 2 hours p.r.n. Most effective when given in small doses at frequent intervals.

B. **Dihydrocodeinone Bitartrate**, N.F. (Hycodan®, Dicodid®): 5-10 mg. ($\frac{1}{12}$-$\frac{1}{6}$ gr.) orally every 3 hours p.r.n. More specific than codeine for cough reflex depression.

C. **Dextromethorphan Hydrobromide**, N.N.D. (Romilar®): 15-30 mg. ($\frac{1}{4}$-$\frac{1}{2}$ gr.) orally 1-4 times daily. 10 mg. ($\frac{1}{6}$ gr.) is equivalent in antitussive effect to 15 mg. ($\frac{1}{4}$ gr.) codeine. Nonnarcotic and nonaddictive.

D. **Noscapine**, N.N.D. (Nectadon®): 15-30 mg. ($\frac{1}{4}$-$\frac{1}{2}$ gr.) orally 1-4 times daily. Nonnarcotic and nonaddictive. Doses as high as 90 mg. (1$\frac{1}{2}$ gr.) daily have been tolerated well for 4-6 weeks.

GASTROINTESTINAL DRUGS

Laxatives and Cathartics.
A. **Lubricants:** Liquid Petrolatum, U.S.P. (mineral oil), or Agar, U.S.P., with mineral oil, 15-45 ml. h.s., p.r.n. Prolonged use may cause loss of fat-soluble vitamins from the gastrointestinal tract.

B. **Saline Cathartics:** Observe caution in the use of sodium salts in cardiac patients.
   1. **Effervescent Sodium Phosphate**, N.F.; Sodium Phosphate, U.S.P. - 5-10 Gm. in a glass of water before breakfast.

Phosphate salts are more palatable than the salts listed below.

2. **Sodium Sulfate, N. F. (Glauber's salts)** - 15 Gm. in a glass of water before breakfast.
3. **Magnesia Magma, U. S. P. (milk of magnesia)** - 15-30 ml. h. s., p. r. n.
4. **Magnesium Sulfate, U. S. P.,** 4-8 Gm. in a glass of warm water before breakfast.

C. Colloid or "Bulk" Laxatives: Psyllium Hydrophyllic Mucilloid, N. N. D. (Metamucil®) - 1-3 tsp. 2-3 times daily p. c. mixed in a full glass of water and followed by a second glass of water.

D. Irritants:
   1. **Aromatic Cascara Sagrada Fluidextract, U. S. P.** - 4-8 ml. h. s., p. r. n. Frequently given in combination with 30 ml. of milk of magnesia or mineral oil.
   2. **Castor Oil, U. S. P.** - 15-60 ml. as a single dose in late afternoon. Capsules and emulsions also available. Emulsions contain 50% of the oil, and twice the dose must be given. Liquid preparation should be mixed with syrup or carbonated beverages to disguise the taste.

E. Newer Laxatives:
   1. **Dioctyl Sodium Sulfosuccinate, U. S. P. (Colace®, Doxinate®)** - 50-100 mg. ($3/4$-$1^1/2$ gr.) 2-3 times daily, or 240-480 mg. (4-8 gr.) once daily. Liquid preparations also available. A surface wetting agent for stool softening. No action on the bowel.
   2. **Bisacodyl (Dulcolax®)** - [Supplied as 5 mg. ($1/12$ gr.) tablets or 10 mg. ($1/6$ gr.) rectal suppositories.] 1-3 (usually 2) tablets h. s., or 1 suppository at the time a bowel movement is desired. Acts by direct contact with the colonic mucosa and stimulates normal peristalsis. Tablets are not to be chewed or taken with antacid.

Antidiarrheal Agents.

A. **Pectin-kaolin Compound (Kaopectate®):** 30-60 ml. q. i. d. (a. c. and h. s.) or after each liquid bowel movement. Often combined in proprietary preparations with antimicrobial agents, e. g., neomycin.

B. **Bismuth Subcarbonate, U. S. P.:** 1-2 Gm. (15-30 gr.) q. i. d. or after each liquid stool.

C. **Bismuth Magma, N. F. (Bismuth Hydroxide and Bismuth Subcarbonate):** 4 ml. q. i. d. or after each liquid bowel movement.

D. **Bismuth Magma and Paregoric:** 4 ml. q. i. d. or after each liquid bowel movement.

E. **Camphorated Opium Tincture, U. S. P. (Paregoric):** 4-8 ml. after each liquid bowel movement p. r. n. Often combined with equal parts bismuth magma. Contraindicated in diarrhea associated with undiagnosed abdominal pain.

F. **Codeine Phosphate, U. S. P.:** 15-65 mg. ($1/4$-1 gr.) subcut. after each liquid bowel movement p. r. n. Contraindicated in diarrhea associated with undiagnosed abdominal pain.

Antiemetics.

A. Triflupromazine Hydrochloride, N.N.D. (Vesprin®): 10-20 mg. ($1/6$-$1/3$ gr.) orally, then 10 mg. ($1/6$ gr.) t.i.d.; 2-10 mg. ($1/30$-$1/6$ gr.) (avg. 8 mg.) I.V.; 5-15 mg. ($1/12$-$1/4$ gr.) (avg. 15 mg.) I.M. Said to be the most potent antiemetic available. Minimum tendency to produce oversedation.

B. Chlorpromazine Hydrochloride, U.S.P. (Thorazine®): 10-50 mg. ($1/6$-$3/4$ gr.) (avg. 25 mg.) orally every 4-6 hours p.r.n.; 25-50 mg. ($3/8$-$3/4$ gr.) I.M. every 4-6 hours p.r.n. See also p. 622.

C. Dimenhydrinate, U.S.P. (Dramamine®): 50-100 mg. ($3/4$-$1\frac{1}{2}$ gr.) (avg. 50 mg.) orally q.i.d. Available also as rectal suppositories, 100 mg. ($1\frac{1}{2}$ gr.) for insertion once nightly. Less effective in prevention of vomiting than phenothiazine derivatives such as chlorpromazine (Thorazine®).

D. Meclizine Hydrochloride, N.N.D. (Bonamine®): 25-50 mg. ($3/8$-$3/4$ gr.) every 6-12 hours. May be taken 1 hour before travel or radiation as antiemetic prophylaxis.

Antacids.

A. Absorbable Antacids: Sodium Bicarbonate, U.S.P., 1 Gm. (15 gr.) in one-half glass of water.

B. Nonabsorbable Antacids: Aluminum Hydroxide Gel, U.S.P., and Magnesium Trisilicate, U.S.P., alone or in combination, liquid or tablets, 4-8 ml. or 1-2 tablets (0.5 Gm. each) with one-half glass of water every 2-4 hours p.r.n., preferably 30-90 minutes before meals.

Anticholinergic Drugs.

A. Belladonna Alkaloids:

1. Belladonna Tincture, U.S.P., 0.3-0.6 ml. (5-10 drops) in one-half glass of water orally, t.i.d., 30 minutes before meals and at bedtime p.r.n. (0.6 ml. of the tincture equals about 0.2 mg. of atropine). Available with phenobarbital.

2. Belladonna Extract, U.S.P., 8-15 mg. ($1/8$-$1/4$ gr.), tablets or capsules, orally t.i.d., 30 minutes before meals and at bedtime p.r.n. (15 mg. equals about 0.2 mg. of atropine alkaloid). Available with phenobarbital.

3. Atropine Sulfate Tablets, U.S.P., 0.3-0.6 mg. ($1/200$-$1/100$ gr.) may be administered orally or subcut. The usual dose is 0.3 mg. ($1/200$ gr.) t.i.d.

B. Synthetic Anticholinergic Drugs: A few are listed below.

1. Methantheline Bromide, U.S.P. (Banthine®), 50-100 mg. ($3/4$-$1\frac{1}{2}$ gr.) every 6 hours around the clock as initial dosage, reduced to one-half for maintenance. Ampuls of 50 mg. are available for I.V. or I.M. use.

2. Propantheline Bromide, N.N.D. (Pro-Banthine®), 15 mg. ($1/4$ gr.) with each meal and 30 mg. ($1/2$ gr.) at bedtime. Available in parenteral form for I.V. or I.M. use.

3. Tridihexethyl Chloride, N.N.D. (Pathilon®), 25 mg. ($3/8$ gr.) t.i.d. before meals and 50 mg. ($3/4$ gr.) at bedtime. Parenteral dose (I.V., I.M., or subcut.) is 10-20 mg. ($1/6$-$1/3$ gr.) every 6 hours.

ANTIMICROBIAL SPECTRA OF CHEMOTHERAPEUTIC AGENTS

The following data are based on clinical and laboratory studies and should be used only as a guide to the selection of drugs when sensitivity tests are not available or until they are completed. There are significant variations in the susceptibility of strains.

Very effective
Moderately effective
Slightly effective

C = Use only in combination with another drug
U = For urinary tract infection only

Organism (Gram Reaction Indicated in Parentheses)	Penicillin	Streptomycin	Tetracyclines	Chloramphenicol	Erythromycin	Novobiocin	Neomycin*	Polymyxin*	Bacitracin*	Sulfonamides	Nitrofurantoin
Staph. aureus(+)	++C	+	++	++	+++C	+++C	+++		+++	+	++U
Staph. albus(+)	++C	+	++	++	+++C	+++C	+++		+++	+	++U
Str. hemolyticus(+)	+++	+	++	++	+++C		+++		+++	+	
Str viridans(+)	++	+	++	++	++		+++C		+++		
Str faecalis(+)	++C	++C	+C	++C	++C		+++		+++		
Esch. coli(-)		++	++	+++			+++	++		++	++U
A. aerogenes(-)		++	++	++			+++	++		+	
P. vulgaris(-)		++	+U	+U		+++C	+++			+C, U	+++U
Ps. aeruginosa(-)		+	+U	+U			++	+++			+++U
Clostridium group (+)	++C		++C	++C			+++			+C	
C. diphtheriae (+)	++		++	++			++				
B. anthracis(+)	++C	++	++	++					+	+C	
A. bovis(+)	+++C	+	+					+C		++C	++
D. pneumoniae(+)	+++		+++	+++	+++C				+++	+	
K. pneumoniae(-)		+++	++	++			++	+++		+	
H. influenzae(-)		+++C	++C	+++C	+			++	+++	++C	
Vincent's bacteroides(-)	++										
Myco. tbc. (+)†		+++C									
N. gonorrhoeae(-)	+++	+	+++	+++	+++C					++	
H. ducreyi(-)		+	+++	++				+	++	+	
Donovania granulomatis		+	++	++							
Lymphopathia ven. virus	+		+++	+++	++C					+	
T. pallidum	+++		++	++	+C		+		+		
T. pertenue	+++		+	+	+		+		+		•
L. icterohaemorrhagiae	+	+	++	+	+C		+				
S. typhi (-)			+	+++			+				
S. paratyphi (-)			+	+++			+			+	
Shigella gr. (-)		+	+++	+++				++		++	
Brucella gr. (-)		++C	++C	+C	+C		+	+		+C	
P. tularensis(-)		+++	+++	+++			+			+	
N. meningitidis(-)	++		++	++						+++C	
Ery. rhusiopathiae	+++	+	+								

*These drugs are nephrotoxic when used systemically. They should be administered only to hospitalized patients who do not respond to less toxic agents.

†Streptomycin (or dihydrostreptomycin) should be used only in combination with another drug such as isoniazid (INH) or aminosalicylic acid (PAS).

ANTIBIOTIC AND CHEMOTHERAPEUTIC DRUGS

Drug	Dosage	Usage	Toxicity
Aminosalicylic Acid, U.S.P. (PAS)	Oral: 3-4 Gm. q. 6 h. I.V.: 15 Gm./day in 5% solution in 2 doses 4 hours apart. 5 mg. of heparin should be added to each L.	Used in the treatment of tuberculosis in combination with more potent agents, particularly streptomycin and isoniazid, to delay emergence of resistant bacilli.	Nausea, vomiting, diarrhea, drug fever, dermatitis, crystalluria, hematuria, and hypoprothrombinemia. Gastrointestinal symptoms can be avoided by parenteral administration of sodium PAS.
Bacitracin, U.S.P.	Oral: 60,000-180,000 U. daily for 20 days in amebiasis: 120,000 U. daily together with polymyxin or neomycin for preoperative preparation of colon. I.M.: 2500-20,000 U. q. 6 h. Topical: In surgical and dermatologic infections in solutions or ointments of 500-1000 U./ml.	Bacitracin has a principally bactericidal action against gram-positive cocci (including penicillin-resistant strains), spirochetes, and Entamoeba histolytica. It is synergistic with penicillin and other drugs in some instances. It is most commonly used topically, but may be useful I. M. in severe systemic infections with certain strains of organisms resistant to other drugs and in combined therapy.	Bacitracin is too toxic for general systemic use. Nephrotoxicity is marked. Albuminuria, cylindruria, and nitrogen retention occur frequently. Daily urine examinations are in order, and the patient should usually be hospitalized during systemic treatment.
Chloramphenicol, U.S.P. (Chloromycetin)	Oral: 0.5 Gm. q. 6 h. I.M. or I.V.: 0.5 Gm. q. 6 h.	Drug of choice in salmonella infections. Inhibits the growth of most gram-negative bacilli and rickettsiae and is effective against some penicillin-resistant gram-positive cocci. It is also useful in selected gram-positive and gram-negative bacterial infections in patients who are sensitive to penicillin and tetracyclines.	Occasionally nausea, vomiting, and diarrhea. Because of its nitro-benzene ring, it is potentially toxic to bone marrow and may rarely produce aplastic anemia or granulocytopenia. Hgb., WBC, and differential should be done every 3 days during treatment.
Erythromycin, U.S.P. (Erythrocin, Ilotycin)	Oral: 0.2-0.4 Gm. q. 4-6 h. in enteric-coated tablets. I.V.: 0.5-1 Gm. q. 8-12 h. in severely ill patients.	Active against most strains of gram-positive cocci, gram-negative cocci, C. diphtheriae, H. influenzae, B. pertussis, and brucellae. May be used as alternative to penicillin in these infections.	Nausea, vomiting, moniliasis infection, and drug fever occur infrequently.

Drug	Dosage	Remarks	Toxicity
Isoniazid, U.S.P. (INH, Nydrazid®)	Oral: 5-10 mg./Kg./day in 2-3 doses; 10 mg./Kg./day should be used in tuberculous meningitis. I.M.: Sterile solutions are available.	Because of rapid emergence of resistant strains, it is usually administered in combination with another antibiotic, especially streptomycin or bacitracin. It should not be used alone in serious staphylococcal infections.	Constipation, dysuria, hyperreflexia, postural hypotension and dizziness, eosinophilia, slight anemia, occasional casts and traces of albumin in the urine, reducing substances in the urine. Toxic psychoses, peripheral neuritis, and leukopenia have been reported, especially with high doses.
Mandelic Acid Salts	Methenamine Mandelate, U.S.P. (Mandelamine®), 3 Gm. (45 gr.) daily by mouth. Ammonium mandelate, 8-12 Gm. daily by mouth. Children: Syrup of ammonium mandelate, 2-4 ml. q.i.d. for a five-year-old child.	These are primarily urinary antiseptics which are excreted in the urine after oral administration in therapeutic concentration but do not produce significant tissue levels. Therefore, a focus of pyelonephritis in the kidney will rarely be affected. They greatly reduce the bacterial count in the urine and thereby diminish symptoms.	None significant.
Neomycin Sulfate, U.S.P. (Mycifradin®)	Oral: 1 Gm. q. 4-6 h. for 2-3 days for preoperative preparation of the intestinal tract. In combination with polymyxin or bacitracin, smaller doses (0.5 Gm. q.6-12 h.) are equally effective. I.M.: 0.5 Gm. q.6-8 h. Rarely used because of toxicity. Topical: 1-10 mg./ml. or 1-5 mg./Gm. in solution or ointments.	Principal use is for bowel preparation and as a topical agent. Neomycin is most active against gram-negative rods, but is also effective against many strains of gram-positive cocci, particularly staphylococci, as well as gram-positive rods. Parenteral administration may rarely be indicated in proteus or staphylococcus sepsis or severe pyelonephritis.	When given parenterally it may cause renal damage or irreversible deafness. Hospitalization during administration is indicated. Intraperitoneal use is dangerous and may cause respiratory paralysis. Oral and topical use is harmless due to poor absorption.

Drug	Dosage	Usage	Toxicity
Nitrofurantoin, N.N.D. (Furadantin⊕)	Oral: 100 mg. q.i.d.	Active against a wide variety of bacteria, both gram-positive and gram-negative. Readily absorbed from the gastrointestinal tract and excreted in high concentration in the urine. Serum and tissue concentrations are insignificant. Chiefly useful for treatment of urinary tract infections where significant tissue invasion and bacteremia do not exist.	Malaise, nausea, skin eruptions. Maximal continuous course is 14 days.
Novobiocin, Sodium or Calcium, N.N.D. (Albamycin⊕, Cathomycin⊕)	Oral: 0.5 Gm. q. 6-8 h.	Should be reserved for use in conjunction with another antimicrobial drug in the treatment of serious staphylococcal infections. Because resistant variants emerge promptly in most bacterial strains, combined therapy is advisable. Novobiocin is active against gram-positive cocci, including some penicillin-resistant staphylococci and some strains of proteus and other gram-negative bacilli.	Drug fever, pruritic skin eruption, marked eosinophilia, and, rarely, granulocytopenia.
Nystatin, N.N.D. (Mycostatin⊕)	Oral: 500,000 U. 3-6 times daily. Children under 2: 400,000-800,000 U.; over 2: 1-2 million U. in 3 doses. Topical: As vaginal suppositories (100,000 U.) once or twice daily, or as ointment (100,000 U./Gm.).	Nystatin is active against a wide variety of fungi and yeasts. It is poorly absorbed from the gastrointestinal tract, thus acts within the lumen of the bowel or wherever topically applied. It may also be given in combination with tetracycline (Mysteclin⊕) to prevent superinfection with monilia.	Too toxic for parenteral administration. No toxicity has been reported with oral or topical use.
Penicillin G (crystalline)	I.M.: 100,000-5 million U. q. 3-12 h. I.V.: Up to 200 million U. daily in 1000-2000 ml. of saline or 5% dextrose solution to which 10-20 mg. (⅙-⅓ gr.) of heparin can	Penicillin is the drug of choice in most gram-positive bacterial infections (including those due to sensitive strains of staphylococci), pneumococci, hemolytic streptococci, nonhemolytic streptococci, anthrax*, actinomycosis*, diphtheria*,	Fever, rashes, urticaria, arthralgia, or a clinical picture similar to serum sickness may develop in up to 10% of persons. Major and fatal anaphylactic reactions occur. A history of penicillin sensitivity

Drug	Dosage	Uses	Remarks / Toxicity
Penicillin G (procaine)	I.M.: 300,000-3 million U. once or twice daily.	clostridia*, and fusospirochetal diseases, also in gonorrhea, syphilis and yaws. In prophylaxis of hemolytic streptococcal infection, long-acting benzathine penicillin G is used. Oral penicillin V is usually advised only in relatively mild infections Large doses, up to many million units, may be given intrapleurally or intra-articularly When combined with streptomycin, penicillin is effective in most mixed infections due to intestinal flora. Topical application is contraindicated.	be added to prevent thrombophlebitis. generally contraindicates its use. Prophylactic administration of corticotropin or corticosteroids may prevent reactions in sensitive individuals requiring penicillin (see p. 607). Penicillin O may be substituted satisfactorily in some cases of penicillin G sensitivity.
Benzathine Penicillin G (Bicillin®, Permapen®, Duapen®)	I.M.: 1.2-2.4 million U. q. 3 weeks.		
Penicillin V	Oral: 200,000 U. q. 3 h.		
Polymyxin B Sulfate, U.S.P. (Aerosporin®)	Oral: 15-20 mg./Kg./day for 10 days in acute bacterial dysentery. Same dose in combination with neomycin or bacitracin may be used for 2-3 days for rapid preoperative preparation of the colon. I.M.: 1.5-2.5 mg./Kg./day in 3 or 4 doses with 1% procaine. Topical: Solutions containing 1 mg./ml. of saline applied to wounds and tracts infected with Pseudomonas aeruginosa.	Drug of choice in serious infections with Pseudomonas aeruginosa (especially bacteremia and meningitis) and other resistant strains of the gram-negative rods. Topically and orally it is not absorbed, but it is effective locally in infected wounds and, when given by mouth, is useful in the treatment of the shigella carrier state and in preoperative bowel preparation.	Most toxic effects occur at dosage levels over 2.5 mg./Kg./day I.M. and tend to be transient. Paresthesias, dizziness, and incoordination occur frequently, but disappear promptly after the drug has been discontinued. Proteinuria, hematuria, and cylindruria may occur, and nitrogen retention is frequent in individuals with impaired kidney function. Hospitalization is required during parenteral therapy.
Streptomycin Sulfate and Calcium Chloride, U.S.P., and Dihydrostreptomycin Sulfate, U.S.P.	Oral: 1 Gm. q. 6 h. for 48-72 h. (For bowel preparation.) I.M.: (1) Nontuberculous infections - 1-4 Gm. (avg. . 1-2 Gm.) daily in divided doses q. 4-8 h., usually	The antimicrobial effects of the two drugs are identical, but streptomycin is preferred because the neurotoxicity (deafness) of dihydrostreptomycin is unpredictable, irreversible, and may be delayed.	Following parenteral administration, rash, eosinophilia, nausea, proteinuria, cylindruria, or nitrogen retention may occur. The most serious effects are on the eighth cranial nerve. Streptomycin may damage the

*Combined therapy.

[Cont'd.]

[Streptomycin and Dihydrostreptomycin, Cont'd.]

Drug	Dosage	Usage	Toxicity
	for not longer than 5 days because resistant strains emerge rapidly. Keep urine alkaline in urinary tract infections. (2) Tuberculous infections - 1 Gm. daily or twice weekly in combination with isoniazid (INH), 5 mg./Kg./day, or aminosalicylic acid (PAS), 10-14 Gm./day, or both.	Streptomycin is the drug of choice in the treatment of tuberculosis, tularemia, urinary tract infections due to sensitive gram-negative bacilli, combined with sulfadiazine as treatment of H. influenzae, K. pneumoniae, and P. pestis infections, and combined with penicillin in mixed infections of the peritoneum. These drugs are not absorbed from the gastrointestinal tract but will eradicate colonic flora if given orally after thorough purgation.	vestibular portion (causing vertigo or ataxia), dihydrostreptomycin the auditory portion (causing deafness).
Sulfadiazine, U.S.P. Sulfamerazine, U.S.P., or Sulfisoxazole, U.S.P. (Gantrisin ®) Sulfonamide Mixtures: (1) Sulfadiazine and sulfamerazine (2) Sulfamerazine, sulfadiazine, and sulfathiazole	Oral: 2-4 Gm. initially, then 1 Gm., q. 4-6 h. In urinary tract infections, the dosage is 0.5 Gm. q. 4-6 h. I.V.: 3-5 Gm. of sodium salt in saline, dextrose, M/6 sodium lactate, or other diluent. Determine maintenance dose with blood level measurement.	Sulfonamides are the drugs of choice in meningococcal infections. They are also useful against dysentery bacilli (shigellae) and in some urinary tract infections with gram-negative bacilli (especially sulfisoxazole, which is more soluble than the others). Effective blood levels are 8-12 mg./100 ml. Blood levels should be determined as a guide to therapy in renal insufficiency and in critical cases. Owing to its greater solubility, sulfisoxazole gives plasma concentrations 3 times as high as can be obtained with other sulfonamides on the same dosage.	Fever, nausea, vomiting, and urticara are commonest. Others include stomatitis, conjunctivitis, arthritis, diarrhea, rashes, periarteritis nodosa, anemia, agranulocytosis, hepatitis, exfoliative dermatitis, oliguria, hematuria, and lower nephron nephrosis. To detect reactions do Hgb., WBC, and differential, and urinalysis for pH and sediment (especially RBC and crystals) every other day. Keep urine output at about 1500 ml./day by sufficient fluid intake and, in intensive therapy, alkalinize the urine by giving sodium bicarbonate, 5-15 Gm. daily.
Succinylsulfathiazole, U.S.P. (Sulfasuxidine ®) or Phthalylsulfathiazole, U.S.P. (Sulfathalidine ®)	For suppression of intestinal flora in preoperative preparation, 0.1-0.3 Gm./Kg./day orally divided into equal doses q. 4-6 h. for 4-5 days preoperatively.	Succinylsulfathiazole and phthalylsulfathiazole are very poorly absorbed from the intestinal tract and are therefore used only for bowel preparation.	

Chlortetracycline, U.S.P. (Aureomycin®) Oxytetracycline, U.S.P. (Terramycin®) Tetracycline, U.S.P. (Achromycin®, Tetracyn®, Polycycline®, Steclin®, Panmycin®)	Oral: 0.25-1 Gm. q. 6 h. I.M.: Oxytetracycline and tetracycline, 0.1 Gm. q. 8 h. Chlortetracycline, 0.25 Gm. in 1% procaine solution with 250 units of hyaluronidase q. 6 h. I.V.: 0.5-1 Gm. q. 12 h	The most important use of the tetra-cyclines is in the treatment of gram-negative bacillary infections and gram-positive infections resistant to penicil-lin. Because of their broad spectrum, they are also widely useful as empirical treatment when bacteriologic diagnosis is unknown, as a supplement to penicil-lin and streptomycin in combined ther-apy, and as an alternative drug in appro-priate penicillin-sensitive patients. The diseases in which tetracyclines are generally applicable include. (1) All mixed bacterial infections caused by sus-ceptible organisms, e. g., peritonitis, urinary tract infections, wound infec-tions, gastrointestinal and respiratory tract infections, (2) penicillin-resistant, tetracycline-sensitive staphylococcal infections; and (3) rickettsial diseases, psittacosis, lymphogranuloma venereum, primary atypical pneumonia, brucellosis.	Vomiting, diarrhea, stomatitis, glossitis, and overgrowth of resis-tant organisms such as monilia, Staphylococcus aureus, pseudo-monas, and proteus; pruritus of the anus, scrotum, or vagina may oc-cur. When monilia are the cause of pruritus or gastrointestinal symp-toms, nystatin (Mycostatin®) should be used. Mysteclin® is a proprie-tary mixture of tetracycline and nystatin. Superinfection of the colon with tetracycline-resistant bacteria may result in a severe form of pseudomembranous colitis.
Tyrothricin, U.S.P. (Soluthricin®)	Topical: Solutions and ointments of 0.5 and 2.5 mg./ml.	Active only against gram-positive organisms. Because of its toxic effects on parenteral administration, this drug is limited entirely to the topical treat-ment of infected wounds and pyoderma by application of solutions or ointments.	Harmless on topical use.

PEDIATRIC DOSAGES OF ANTIBIOTIC AND CHEMOTHERAPEUTIC AGENTS

(NOTE: Premature infants and newborns should receive one-half of lower dose of parenteral medication.)

Drug	Oral	Intramuscular	Intravenous	Intrapleural Intra-articular Intraperitoneal
Penicillin	100,000-300,000 U. (penicillin G, buffered, or penicillin V) 5 times daily, 30 min. a. c.	10,000 U./lb. or 100,000-300,000 U./day	10,000 U./lb./day	10,000-20,000 U./ml.
Benzathine Penicillin G (Bicillin®, Permapen®, Duapen®)	300,000 U. q.i.d.	600,000 U. or 1,200,000 U. as single injection		
Streptomycin, U.S.P.	5 mg./lb. q.i.d.	5-10 mg./lb. b.i.d.		50 mg./ml.
Tetracyclines [Achromycin®, Tetracyn®, Panmycin®, Polycycline®, Aureomycin® (1), Terramycin® (2)]	2.5-5 mg./lb. q.i.d.	3 mg./lb. b.i.d.	3 mg./lb. b.i.d. (1 mg./ml.)	5 mg./ml.
Chloramphenicol, U.S.P. (3) (Chloromycetin®)	5-15 mg./lb. q.i.d.	15-30 mg./lb. b.i.d.	7.5-15 mg./lb. b.i.d. (5 mg./ml.)	5 mg./ml.
Erythromycin, U.S.P. [Erythrocin®, Ilotycin®, Ilosone® (4)]	3.75-7.5 mg./lb. q.i.d.		8 mg./lb. b.i.d. (0.5-1 mg./ml.)	1 mg./ml.
Novobiocin, U.S.P. (Cathomycin®, Albamycin®)	2.5-5 mg./lb. q.i.d.	4-7.5 mg./lb. b.i.d.	4-7.5 mg./lb. b.i.d.	
Kanamycin, N.N.D. (Kantrex®)	12.5 mg./lb. q.i.d.	1-3.5 mg./lb. b.i.d.		2.5 mg./ml.
Neomycin, U.S.P. (Mycifradin®)	12.5 mg./lb. q.i.d.	1.3 mg./lb. t.i.d.		2 mg./ml.
Polymyxin, U.S.P. (Aerosporin®)	1.25-2.5 mg./lb. q.i.d.	0.3 mg./lb. t.i.d.		1 mg./ml.

	250 U./lb. q.i.d.	100-200 U./lb. t.i.d. Never over 50,000 U. per day in 3 doses.		1000 U./ml.
Bacitracin, U.S.P.				
Sulfisomazole, U.S.P. [Gantrisin® (5)]	15 mg./lb. q.i.d.	20 mg./lb. t.i.d.	20 mg./lb. t.i.d. (50 mg./ml.)	
Sulfadiazine, U.S.P., and Sulfamerazine, U.S.P. Sodium salts (5)	15 mg./lb. q.i.d.		20 mg./lb. t.i.d. (50 mg./ml.)	
Succinylsulfathiazole [Sulfasuxidine (5)]	25 mg./lb. q.i.d.			
Thiazosulfone, N.N.D. [Promizole®]	0.125-1.25 Gm. q.i.d.			
Nitrofurantoin, N.N.D. [Furadantin®]	0.75-1.25 mg./lb. q.i.d.			
Isoniazid, U.S.P. [INH, Nydrazid®]	4 mg./lb. b.i.d.	2.5 mg./lb. b.i.d.		
Aminosalicylic Acid, U.S.P. [PAS]	0.5-2 Gm. q.i.d.		0.5-2 Gm. q.i.d. (30 mg./ml.)	

(1) Chlortetracycline.
(2) Oxytetracycline.
(3) Should be administered with extreme caution to premature infants.
(4) Ilosone® (propionyl erythromycin ester): Oral dosage only.
(5) Initial dose should be twice subsequent dose.

TRANQUILIZER DRUGS AND THEIR USE IN ANESTHESIA

	Adult Dosage	Duration of Action	Use in Anesthesia	Complications
Reserpine, N.N.D. (Serpasil®, Reserpoid®, etc.)	For anxiety: 0.1-0.25 mg. (1/600-1/250 gr.) orally daily to t.i.d. For hypertension: 0.25 mg. (1/250 gr.) t.i.d., and gradually reduce to maintenance dose of 1-2 times daily.	Sedation: 6 hours. Hypertension: 1-2 weeks.	Not useful for premedication due to delayed action.	Hypotension. No "epinephrine inversion" (see Chlorpromazine, below), but requires larger doses of vasopressor to reverse hypotension.
Meprobamate, N.N.D. (Miltown®, Equanil®, etc.)	200-800 mg. (3-12 gr.) orally 3-4 times daily.	4-6 hours.	400-800 mg. (6-12 gr.) as hypnotic premedication.	Skin allergies. No sympatholytic or ganglionic blocking action. No "epinephrine inversion" (see Chlorpromazine, below). Does not cause hypotension. Weak central action.
Chlorpromazine, N.N.D. (Thorazine®)	Tranquilizer, antiemetic: 10-50 mg. (1/6-3/4 gr.) orally or I.M. 3-4 times daily. Premedication: 50-100 mg. (3/4-1 1/2 gr.) orally; 25 mg. (3/8 gr.) I.M.; 5-10 mg. (1/12-1/6 gr.) I.V.	6-8 hours.	Premedicant. Antiemetic. Sympatholytic. Depresses cardiorespiratory reflexes. Prevents myocardial irritability. Facilitates hypotension and hypothermia.	Hypotension; when treated by epinephrine may lead to "epinephrine inversion" and more severe hypotension. Recommend first norepinephrine, then phenylephrine.
Promethazine HCl, N.F. (Phenergan®)	Sedation (every 4-6 hours): 25-50 mg. (3/8-3/4 gr.) orally; 15-25 mg. (1/4-3/8 gr.) I.M.; 8-15 mg. (1/8-1/4 gr.) I.V. Premedication is twice above dosage.	4-6 hours.	Antiemetic, premedication.	Hypotension rare in therapeutic doses. No depression of cardiorespiratory reflexes. Useful sedation for regional anesthesia.
Promazine HCl, N.N.D. (Sparine®)	Tranquilizer (every 4-6 hours): 25-50 mg. (3/8-3/4 gr.) orally; 15-25 mg. (1/4-3/8 gr.) I.M.; 8-15 mg. (1/8-1/4 gr.) I.V. Premedication is twice above dosage.	4-6 hours.	Parasympatholytic premedication. Action essentially the same as but milder than chlorpromazine.	Drowsiness, hypotension, dizziness.

VASOPRESSORS AND THEIR USE IN ANESTHESIA*

	Levarterenol Bitartrate, U.S.P. (Levophed®)	Ephedrine Hydrochloride, N.F., or Sulfate, U.S.P.	Methoxamine Hydrochloride, U.S.P. (Vasoxyl®)	Metaraminol Bitartrate, N.N.D. (Aramine®)	Phenylephrine Hydrochloride, U.S.P. (Neo-Synephrine®)
Dosage and Duration					
Intramuscular	May cause tissue slough	20-50 mg. 30-40 min.	10-20 mg. 30-60 min.	2-10 mg. 20-60 min.	2-5 mg. 30-60 min.
Intravenous single dose		10-25 mg. 20 min.	4-10 mg. 10-30 min.	0.5-2 mg. 20-60 min.	0.25-0.50 mg. 20 min.
Intravenous continuous drip	Usual method, 4 mg./1000 ml.	Not usually employed	Not usually employed	May be used	1-2 mg./100 ml.
Effects					
Myocardial stimulation	Yes	Yes	No	Yes	
CNS stimulation	Slight	Marked	None	Slight	Slight
Cardiac rate	Increased or decreased	Increased	Reflex slowing effect	Increased, or reflex slowing effect	Reflex slowing effect
Peripheral vessels	Constricts	Constricts	Constricts	Constricts	Constricts
Use with cyclopropane, chloroform, trichloroethylene, halothane	Dangerous, may cause ventricular fibrillation	Yes, but causes supraventricular arrhythmias	Yes		Yes

*Epinephrine is not employed during anesthesia as a systemic vasopressor.

MUSCLE RELAXANTS COMMONLY USED IN ANESTHESIA

	Tubocurarine Chloride, U.S.P. (Tubarine®)	Gallamine Triethiodide, N.N.D (Flaxedil®)	Decamethonium Bromide, N.N.D. (Syncurine®)	Succinylcholine Chloride, U.S.P. (Anectine®, Quelicin®, Sucostrin®)
How supplied	10 ml. vials, 3 mg. (1/20 gr.)/ml.	10 ml. vials, 20 mg. (1/3 gr.)/ml.	10 ml. vials, 1 mg. (1/60 gr.)/ml.	10 ml. vials, 20 mg. (1/3 gr.)/ml. 10 ml. ampuls, 100 mg. (1 1/2 gr.)/ml.
Dose				
Initial	3-15 mg. (1/20-1/4 gr.) I.V.	40-100 mg. (2/3-1 1/2 gr.) I.V.	1-4 mg. (1/60-1/15 gr.) I.V.	20-60 mg. (1/3-1 gr.) I.V.
Subsequent	1/2-1/3 previous I.V. dose	1/3 previous I.V. dose	0.5-1 mg. I.V.	Same as initial I.V.
Continuous drip	Not used	Not used	Not used	0.1-0.2% solution
Onset of action	3-5 minutes	2-5 minutes	3-5 minutes	1-3 minutes
Duration of action of single paralyzing dose	30-60 minutes	25-30 minutes	20 minutes	3-5 minutes
Side effects	Histamine release, hypotension.	No histamine effect; tachycardia.	May have tachyphylaxis. Effect erratic.	Muscle fasciculations with rapid injection. Salivation. Increases intraocular tension.
Other effects	Cumulative action. Additive with ether.	Cumulative action. Additive with ether.	Usually no cumulative effect. Not additive with ether.	Not additive with ether. Bradycardia. May produce anti-depolarizing type block.
Contraindications	Myasthenia gravis.	Myasthenia gravis.		Severe liver disease. Increased intraocular tension.
Antidotes*	(1) Edrophonium Chloride, N.N.D. (Tensilon®): 10-20 mg. (1/6-1/3 gr.) I.V., may be repeated. (2) Neostigmine, U.S.P. (Prostigmin®): 0.5-2 mg. (1/120-1/30 gr.) I.V.; precede with Atropine Sulfate, U.S.P., 0.4-0.6 mg. (1/150-1/100 gr.) I.V.	Same as Tubocurarine.	None	After large total dosage a prolonged apnea may be reversed by neostigmine (Prostigmin®). Transfusion.

*Do not use antidotes for muscle relaxants unless the patient shows evidence of spontaneous respiration.

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*Individual drugs are not listed in the Index. See Appendix; Drug Dosages, pp. 805-824.

Method A Clear mouth and throat Place patient supine Insert left thumb between patient s teeth grasp mandible firmly at midline and draw it forward (upward) so that the lower teeth are leading Close patient s nose with right hand Gauze (as shown) or airway (see below) may be used but are not necessary. See also p 7

Method B Clear mouth and throat Place patient supine Pull strongly forward at angle of mandible Close patient s nose with your cheek Gauze (as shown) or airway (see below) may be used but are not necessary See also p 7
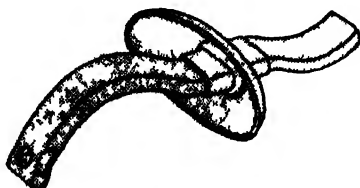
Airway for Use in Mouth-to-Mouth Insufflation The larger airway is for adults The guard is flexible and may be inverted from the position shown for use with infants and children